SIGMUND FREUD

ON APHASIA

Authorized Translation by

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ON APHASIA

ON APHASIA

A Critical Study

by

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Dedicated to Dr. Josef Breuer in friendship and respect.

Authorized Translation with an Introduction
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INTRODUCTION

Freud's book on aphasia is known to a small circle of experts only. Unobtainable for many years it has until recently been regarded as no more than an item on the list of Freud's "pre-psychoanalytical" publications and of no relevance to his later work. Freud himself, having turned to the study of the neuroses, hardly ever referred to his earlier writings. And yet, the period during which they originated was one of intensive and fruitful activity. Not only did Freud make valuable contributions to neurology but he laid the foundations of psychoanalysis. It has gradually been recognized in recent years that his anatomical, neurological and psychoanalytical works form a continuum. The book on aphasia demonstrates this clearly.[1] It

[1] In this introduction I have drawn on the writings of the following authors who discussed the significance of the book from various points of view:

Dorer, M. *Historische Grundlagen der Psychoanalyse* (Historical foundations of psychoanalysis). Leipzig, 1932.

Binswanger, L. *Freud und die Verfassung der klinischen Psychiatrie* (Freud and the Magna Charta of clinical psychiatry). Schweiz. Arch. Neur. Psychiat., Vol. 37, 177, 1936,

Brun, R. *Sigmund Freud's Leistungen auf dem Gebiete der organischen Neurologie* (Freud's achievements in neurology). Schweiz. Arch. Neur. Psychiat., Vol. 37, 199, 1936.

Jelliffe, E. S. *Sigmund Freud as a Neurologist.* J. Nerv. Ment. Dis., Vol. 85, 696, 1937.

Bernfeld, S. *Freud's Earliest Discoveries and the School of Helmholtz.* Psychoan. Quarterly. Vol. 13, 341, 1944.

Idem. *Freud's Scientific Beginnings.* Amer. Imago, Vol. 6, 156, 1949; Yearbook of Psychoanal., Vol. 6, 24, 1950.

Kris, E. "*The Significance of Freud's Earliest Discoveries.*" Internat. J. Psychoan., Vol. 31, 1. 1950.

Riese, W. *Concepts of Evolution and Dissolution of Functions in Psychopathology.* Proc. First Internat. Congress on Psychiatry 1950, Vol. 1, 501, Paris 1952.

Jones, Ernest. *Sigmund Freud: Life and Work*, Vol. 1, London 1953.

I am indebted to Dr. Ernest Jones for the loan of a German copy of Freud's book and of a copy of Dorer's book.

was the first of the author's studies dealing with mental activities and thus provides a link between the two apparently separate periods in his working life. It is worthy of serious attention to-day no less than it was sixty-two years ago. The neurologist will find it not only historically interesting, but also full of stimulating and original ideas concerning problems which are as topical at present as they were then. The psychoanalyst and psychiatrist will recognize it as the most significant forerunner of the author's later work.

As a contribution to the problems of the speech disorders due to brain lesions Freud's treatise stands out among the voluminous writings of the time. It appeared when neurologists were intensely preoccupied with the localization of cerebral functions. The discoveries of Hitzig and Ferrier were fresh in their minds. Broca and Wernicke had established the relationship of certain brain lesions to specific types of aphasia, and the exact localization of all the functions of speech seemed to be within easy reach. Freud was the first in the German speaking world to subject the current theory of localization to a systematic critical analysis. In challenging both a powerful scientific trend and its most influential representatives he showed himself an independent thinker of considerable courage.

Freud had been stimulated to the study of the subject by a paper by Exner and Paneth;[1] this was a report on a piece of experimental research, by which the two physiologists were able to demonstrate that surgical isolation of a cortical area in dogs had the same effect as its excision. They attributed this to two factors: the cutting of the association fibres and "traumatism", thus invoking a localized lesion as well as a functional disturbance. The same combination was considered by Freud to be responsible for the peculiarities of certain types of aphasia. There can be little doubt that his contact with Charcot also contributed to the choice of the subject. But these were only accidental factors; it was almost inevitable for a neurologist with Freud's profound interest in mental processes to be attracted to the study of the aphasias.

Although the book is in many respects a period piece, it still has a message for the neurologist of to-day. Freud's insistence

[1] Exner, S. and Paneth, J. *Über Sehstörungen nach Operationen am Vorderhirn* (Visual disturbances following operations on the telencephalon). Pflüg. Arch., Vol. 40, 62, 1887.

on the compatibility of the functional, i.e., dynamic, with the localizatory approach is still insufficiently heeded by many. In his view on localization he followed Hughlings Jackson. He rejected strict localization not only for the function of speech but also for individual muscles. However, the "speech apparatus" which, although not identical with the structural substrata of speech is in some way related to the latter, is a Freudian concept. The differentiation between a central speech area and the so-called speech centres bordering on the receptive and motor cortical areas functionally related to them, is a most interesting theory which has proved very fruitful.

The proposed division of the aphasias into three groups was a bold attempt at establishing a consistent psychological system based on the theory of associations applied to speech. Considering that the current classification was then, and still is, a confusing mixture of anatomical, physiological and psychological concepts, Freud's system had much to recommend it. However, it was too closely linked to a questionable theoretical framework to be acceptable to the clinicians, though part of it has survived. It was a forerunner of Head's classification which was also based on psychological criteria.

None of the leading authorities in the field of aphasia escaped Freud's criticism, with the notable exception of Hughlings Jackson for whom the author had nothing but praise and whom he pronounced his guiding spirit in the study of the speech disorders. He also appreciated Bastian's contributions, without however accepting his views on physiological speech centres. He quoted Jackson's warning against the confusion of the physical with the psychic and declared himself an adherent of the "Law of Concomitance" adopted by Jackson. He cited some of Jackson's most illustrative examples of "recurrent utterances" in the origin of which emotional factors had played a conspicuous part; in order to underline the importance of these factors in situations of stress he related an interesting self observation.

It is obvious that Hughlings Jackson had made a deep impression on Freud. The following passage (p. 87) shows how fully he had made Jackson's basic doctrine of the evolution and dissolution of function his own: "In assessing the functions of the speech apparatus under pathological conditions we are adopting as a guiding principle Hughlings Jackson's doctrine

that all these modes of reaction represent instances of functional retrogression[1] (disinvolution) of a highly organized apparatus, and therefore correspond to previous states of its functional development. This means that under all circumstances an arrangement of associations which, having been acquired later, belongs to a higher level of functioning, will be lost, while an earlier and simpler one will be preserved. From this point of view a great number of aphasic phenomena can be explained".

Here, then, we find for the first time in Freud's writings the principle of regression which underlies all the genetic propositions of psychoanalysis. Freud had probably come across this principle in some form or other earlier, possibly in Meynert's writings; but nowhere had it been stated so clearly and its applications to psychopathology been pointed out so persistently as in the writings of Hughlings Jackson, who himself had adopted it from Herbert Spencer, the philosopher-psychologist of evolution. The close relationship of psychoanalysis to the theory of evolution was noted by Ernest Jones long ago.[2]

The important role played by Freud's study of the aphasias in the foundation of psychoanalytic theory has been fully recognized by L. Binswanger. He believes that, by acquainting Freud with Hughlings Jackson's genetic doctrine, it had a decisive influence on Freud's thinking, and he goes so far as to state that without knowledge of this book a full historical understanding of Freud's teachings is impossible. A study of the two articles by Hughlings Jackson referred to by Freud will convince the reader that Binswanger's contention is not an overstatement. In these papers Jackson not only applied Spencer's doctrine to the speech disorders but also adumbrated their importance for the study of "insanity". He also expressed the view that certain psychic states and utterances were the results of conflicting nervous discharges. All this must have been of

[1] The term used in the German original is "Rückbildung". The translation of this term chosen is "retrogression" rather than "regression". The latter would be equally correct but has been avoided as it may have created the impression that the German word used by Freud was "Regression". In fact, the latter term was used in "Die Traumdeutung" (1900) for the first time.

[2] Jones, Ernest. Preface to "Papers on Psychoanalysis", London, 1912.

absorbing interest to Freud who was acquainted with concepts of psychodynamics through Herbart, Fechner and Brücke.

The idea that disturbances of function similar to those caused by brain lesions occur in the healthy person under conditions of fatigue and lack of attention, was implicit in the theory of evolution and dissolution. It was to prove of far-reaching importance in psychopathology. It is therefore not surprising to find observations in this book which foreshadowed important psychopathological discoveries. What Freud said about paraphasia, i.e. the mistaken use of words, reads like a prelude to the chapter on errors and slips of the tongue in "Psychopathology of Everyday Life". Freud's observations on paraphasia are still up-to-date. This crucial problem of aphasia has hardly been advanced since.

The "speech apparatus" is the elder brother of the "psychic apparatus" to the working of which most of Freud's later researches were devoted. Both terms obviously have their origin in Meynert's writings. They demonstrate Freud's lasting attachment to physiological concepts.

The book contains a number of other terms which have become household words in psychology and psychiatry. "Projection" and "representation" which were to play such an important part in psychoanalytical theory, are here used in their original sense. The term "Besetzung" and "besetzen" (occupation, occupy; cathexis, cathect) had been used by Meynert for the hypothetical process of the investment of unused cortical cells with new function. Although Freud rejected Meynert's hypothesis he later used these terms for the mechanism of the investment of objects with libido.

The concept of "over-determination" also was defined for the first time in relation to functions of speech which were supposed to be safeguarded against breakdown by a multiplicity of complementary mechanisms.

Freud's preference for concepts implying dynamic processes rather than static conditions is conspicuous throughout the book. It is most clearly expressed in the remarkable passage concerning memories (p. 56). Considerations of this kind must have played their part in the discovery of unconscious mechanisms which was to become Freud's most important contribution to psychology and psychiatry.

The book seems to have received little immediate attention

and its sale was disappointing.[1] The author himself regarded this work with some pride and in one of his letters[2] he spoke of it as something "really good", complaining at the same time that it was hardly taken notice of. This was not surprising; Freud occupied no official position such as was held by those whose theories he criticized so severely. He had not written about aphasia before nor did he pursue the subject. Besides, the book did not contain new clinical observations and was published as a monograph which soon went out of circulation. Possibly the fate of this study would have been different had it been published in one of the leading journals. However, it was not long before the tide of narrow localization theories subsided, and in the first decade of this century Freud's ideas were taken up by some students of aphasia. Storch[3] based his interesting theory of inner language on them. He was followed by Kurt Goldstein[4] who went back to Hughlings Jackson and Freud in evolving the most consistent and fruitful modern concept of aphasia. His differentiation of the central aphasias from the speech disorders due to disturbance of the instrumentalities of language derive directly from Freud. Some other writers also referred to him. The concept of the agnostic aphasias met with considerable interest and the term "agnosia" was generally accepted. Even now Freud's book is quoted with respect in some surveys of aphasia. Thiele[5], in an important monograph, frequently referred to it and remarked that it had remained a work of topical interest even to-day. Nielsen[6] gave it its due place in his historical survey.

The book seems to have made little impression on the French neurologists and it has been unknown to most English and

[1] 142 copies were sold in the first year and 115 in the following nine years. I am indebted for this information to Dr. Ernest Jones.

[2] Freud, Sigm. *Aus den Anfängen der Psychoanalyse*, (The Origins of Psychoanalysis), p. 94, London, 1950.

[3] Storch, E. (1903) *Der aphasische Symptomenkomplex* (The syndrome of aphasia). Monatsch. Psychiatrie & Nervenkrankh., 13.

[4] Goldstein, Kurt. (1912). *Die zentrale Aphasie* (Central aphasia), Neurol. Centralblatt, 12, p.1.

[5] Thiele, W. *Die Aphasien.* Handb. d. Geisteskr., Allgem. Teil, Vol. 2, p. 242. Berlin 1928.

[6] Nielsen, J. M. *Agnosia, Apraxia, Aphasia.* New York & London 1947.

American writers. Jeliffe, and recently Ernest Jones took Head[1] to task for completely ignoring Freud's book when he pronounced his wholesale condemnation of neurologists for their disregard of Hughlings Jackson. Obviously, Head had never read the book, although he quoted Freud as the originator of the term "agnosia". There can be no doubt that at the time of its publication Freud stood alone in his whole-hearted appreciation of Hughlings Jackson. If only for this historical fact, the book deserves to be saved from oblivion. But there is another reason, weightier than considerations of historical justice, which makes it desirable that this book should not remain unknown in the English speaking world: it appears that Freud's direct contact with the evolutionary theories emanating from England was a highly significant event in the development of psychoanalysis. The book bears witness to that encounter.

E. STENGEL

[1] Head, Henry. *Aphasia and Kindred Disorders of Speech*, Vol. 1. Cambridge 1926.

I

The subject I am going to discuss, without presenting new clinical observations, is one to the study of which the best brains of German and foreign neurology have already devoted their efforts (Wernicke, Kussmaul, Lichtheim and Grashey, Hughlings Jackson, Bastian and Ross, Charcot, and others). I therefore propose to state at once which aspects of the problem I hope to advance. I shall endeavour to demonstrate that the theory of aphasia jointly built up by the above-named writers, contains two assumptions which might profitably be revised. The first refers to the differentiation between aphasias caused by destruction of centres and aphasias caused by destruction of pathways. It has been accepted by almost all authors who have written on this subject. The second assumption is concerned with the topographical relationship between the individual speech centres. It was adopted mainly by Wernicke and those workers who have accepted, and enlarged upon, his views. As both hypotheses are important parts of Wernicke's theory of aphasia, my objections to them will take the form of a critique of the theory. They are also intimately related to the idea of "localization", i.e., of the restriction of nervous functions to anatomically definable areas, which pervades the whole of recent neuro-pathology. I shall have to consider the significance of

this factor for the understanding of aphasia in general.

In doing so I have to turn to a famous chapter in the history of the knowledge of the brain. In 1861 Broca[1] presented to the Société Anatomique of Paris the two post-mortem findings which enabled him to conclude that a lesion in the left third frontal convolution caused complete loss or severe reduction of articulate speech whereas the other speech functions and the intellect remained unimpaired. The qualification that this applied to right-handed subjects only, was added later. Broca's discovery has from time to time been disputed. This has no doubt been due to the tendency to reverse Broca's statements and to conclude that loss or impairment of articulate speech necessarily implies the presence of a lesion in the third left frontal convolution. Thirteen years later, Wernicke[2] published the short essay on the symptoms of aphasia which brought him lasting fame. He described another type of speech disorder which forms the counterpart to Broca's aphasia, i.e., loss of understanding with preservation of the ability to use articulate language. He attributed this disorder to a lesion of the first temporal convolution which he had found in his cases. This discovery was bound to give rise to the hope that some day it would be possible to relate the various dissociations of the faculty of speech observed in clinical practice to a corresponding number of well defined cerebral lesions. Wernicke's observations were only the first step towards this aim. He believed that he could see the way from the explanation of aphasia by localized brain lesions to an understanding of the physiological process of speech which appeared to him, in short, as a cerebral reflex: in his view, the speech

[1] P. Broca: *Sur le siège de la faculté du langage articulé avec deux observations d'aphémie (perte de la parole)*, 1861.

[2] Wernicke: *Der aphasische Symptomenkomplex* (The aphasic syndrome), Breslau, 1874.

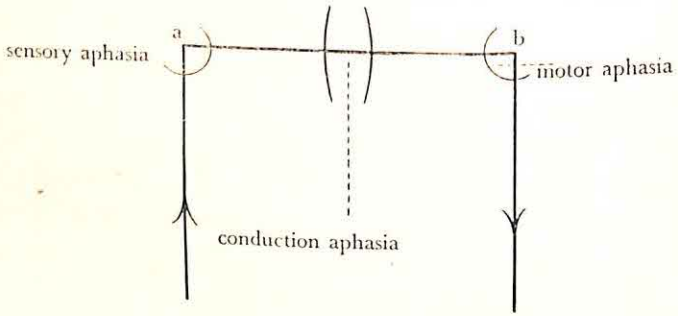
sounds were conveyed via the acoustic nerve to an area in the temporal lobe, the sensory speech centre, whence the stimuli were transmitted to Broca's area, the motor speech centre, which sent the impulse to articulate speech to the periphery.

Wernicke had formed a definite idea as to how the word sounds were contained in the centre. This concept is of fundamental importance for the whole theory of localization. His answer to the question how far psychic functions could be localized was that this was permissible for the most elementary functions only; a visual perception could be related to the cerebral termination of the optic nerve, an auditory perception to the corresponding cortical area of the eighth nerve, etc. Everything beyond this, such as the association of various ideas to a concept, was a function of the association systems connecting different parts of the cortex; they could no longer be localized in one particular area. The sensory stimuli, however, which reached the cerebral cortex, left behind lasting impressions, each of which, according to Wernicke, was stored in a separate cell. "The cerebral cortex with its 600 millions of cells according to Meynert's estimation, offers a sufficiently great number of storage places in which the innumerable sensory impressions provided by the outer world can be stored one by one without interference. The cerebral cortex is populated with such residues of past stimuli which we propose to call memory images."

Such images of the speech sounds are supposed to lie enclosed in the cells of the sensory centre in the first temporal convolution, while Broca's centre contains the images of the speech movements, the "glosso-kinaesthetic impressions". Destruction of the sensory centre causes loss of the sound images resulting in inability to understand language, i.e., sensory aphasia, word deafness.

Destruction of the motor centre eliminates the images of the speech movements, thus making the innervation of the cranial motor nerves for the production of speech sounds impossible, i.e., causing motor aphasia. In addition, the motor and the sensory speech centres are linked to each other by a tract of association fibres which Wernicke, in the light of anatomical studies and of clinical observation, located in the region of the insula. It is not quite clear whether in his view this association is provided by white fibres alone, or also via the grey matter of the insula. He mentions that fibrae propriae originate from the convolutions which encircle the Sylvian fissure. These fibres, he assumes, terminate in the insula which in consequence resembles a big spider concentrating on to itself the fibres converging from all parts of the surrounding cortex. This creates, more than anywhere else in the central nervous system, the impression of a real centre for certain functions. However, the only function attributed to the insula by Wernicke, is that of the association of "word sound image" and "kinaesthetic word impression", both of which are localized elsewhere in the cortex. A function such as this is usually attributed to white fibres only. The destruction of this fibre tract is supposed to cause a speech disorder consisting of paraphasia with normal comprehension and articulation, i.e., confusion of words

FIG. 1



and uncertainty in their use. Wernicke designated this type of speech disorder "conduction aphasia" and differentiated it from the two other "centre aphasias" (Fig. 1).

I am reproducing another drawing from Wernicke's writings in which a schema of the process of speech is superimposed over a diagram of the brain, in order

FIG. 2

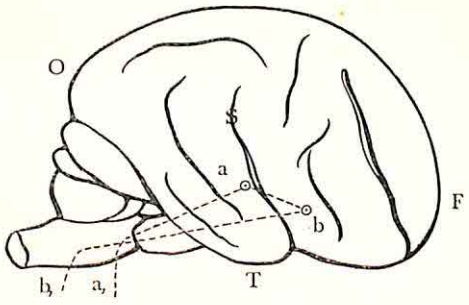


Fig. 3 in *Wernicke:* Der aphasische Symptomenkomplex (The aphasic syndrome)
F, *T*, *O* the frontal, temporal and occipital poles; *S* Sylvian fissure; *a* the central termination of the auditory nerve; a_1 its entrance in the medulla oblongata; *b* area of the kinaesthetic impressions essential to articulate speech; b_1 the centrifugal speech tract emerging from the medulla oblongata.

to show where his schema called for further elaboration: it presents the apparatus of speech without relation to the activities of the rest of the brain and might be applicable to the activity of repeating words heard. But if one takes into account the various other activities of the speech centres which are indispensable for spontaneous speech, a more complicated presentation of the speech apparatus is called for, which would hold out the prospect of explaining a greater number of speech disorders by localized lesions. Lichtheim[1] (1884) took this step, and in a consistent elaboration of Wernicke's

[1] Lichtheim: *Ueber Aphasie* (On Aphasia). Deutsch. Arch. f. klin. Med., Vol. 36; *On Aphasia.* Brain, Jan. 1885.

FIG. 3

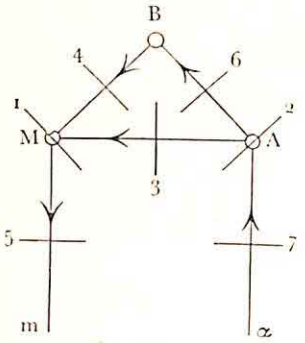


Fig. 1 in *Lichtheim:* On Aphasia. "Brain", VII, p. 436.

approach he arrived at the schema of the apparatus of speech which I am reproducing here (Fig. 3).

In this schema, M stands for the motor speech centre (Broca's area). 1 signifies the motor aphasia caused by its destruction. A represents the auditory speech area, 2 the auditory (sensory) aphasia resulting from its destruction. 3, 4, 5, 6 and 7 signify conduction aphasias.[1] 3 indicates the conduction aphasia of the insula postulated by Wernicke. Point B has not the same significance in the schema as A and M which correspond to circumscribed areas of the cerebral cortex. B is no more than a schematic representation of the numerous parts of the cortex from which the speech apparatus can be stimulated into action. For this reason a speech disorder due to lesion of that point does not appear in the schema.

Lichtheim divided the seven types of aphasia postulated in his schema into centre aphasias (1, 2), peripheral conduction aphasias (5, 7) and central conduction aphasias (3, 4, 6). Wernicke[2] later replaced this nomenclature by another which, although not without faults, has the advantage of having been generally accepted. According to the latter, Lichtheim's seven forms of aphasia are to be designated and defined as follows.

1. *The cortical motor aphasia.* Understanding of spoken

[1] The English term proposed by Lichtheim for this type of speech disorder was "commissural aphasia", but the term "conduction aphasia" used by Head and later writers is preferable (Transl.).

[2] Wernicke: *Die neueren Arbeiten über Aphasie* (Recent studies on aphasia). Fortschritte d. Medicin, 1885, p. 824; 1886, p. 371, 463.

language is intact, but the vocabulary is lost, or limited to a few words. Spontaneous speech and repetition of words heard are both impossible. This form is identical with Broca's well-known aphasia.

5. *The subcortical motor aphasia.* This differs from the above-named in one respect, i.e., by the preservation of the ability to write, and, allegedly, by another peculiarity to be mentioned later.

4. *The transcortical motor aphasia.* In this form spontaneous speech is impaired, but the ability to repeat words heard is preserved, which results in a peculiar dissociation of the motor component of speech.

2. *The cortical sensory aphasia* (Wernicke's aphasia). The patient fails to understand spoken language, nor is he able to repeat it, but he can speak spontaneously with an unlimited vocabulary. However, his spontaneous speech is not intact but shows "paraphasia". This feature, which is of far-reaching importance, will be discussed later.

7. *The subcortical sensory aphasia*, which differs from (6) by the absence of paraphasia in spontaneous speech.

6. *Transcortical sensory aphasia.* This form presents the most unexpected dissociation of speech, but one that follows of necessity from Lichtheim's schema. The patient's spontaneous speech is paraphasic, he is capable of repeating but unable to understand what he is told and what he has repeated.

3. *Wernicke's conduction aphasia*, characterized by paraphasia in the absence of other symptoms.

I am reproducing another schema by which Lichtheim attempts to account for the impairment of written language by postulating a visual and a writing centre with their respective connections (Fig. 4). However, it fell to Wernicke,[1] who closely followed Lichtheim's

[1] Wernicke, l. c.

FIG. 4

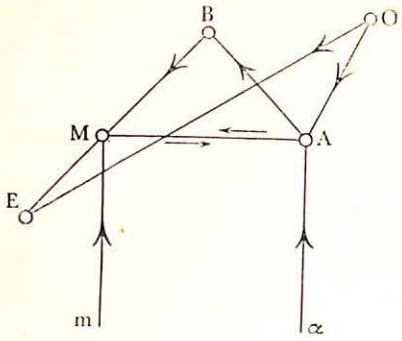


Fig. 2 in *Lichtheim:* On Aphasia. "Brain", p. 437. In this schema *O* signifies the centre for visual impressions, *E* the centre for cheiro-kinaesthetic impressions. In the same article (p. 443) Lichtheim presents another schema in which *E* is directly connected with *A* and *O* instead of with *M* and *O*.

procedure, to complete this task in a later publication.

Lichtheim is said to have corroborated all forms of dissociation of speech function arising from his schema by cases which he actually observed clinically though their number was small. This makes the great success of his theory of aphasia appear well deserved. Lichtheim's schema which had been evolved by way of deduction, anticipated unexpected and hitherto unobserved forms of speech dissociation. If these postulated forms could be confirmed by clinical observation, this was bound to appear as valid proof for the correctness of Lichtheim's premises. It is not a reproach against Lichtheim to point out that his schema must not be understood in the same way as Wernicke's. The latter can, as it were, be inscribed into the brain, as the localization of the centres and fibre tracts which it contains has been anatomically verified. Lichtheim's schema, however, postulates new tracts, the knowledge of which is still lacking. It is therefore impossible to say whether his centres and fibre tracts are related in space to each other in the way they are presented in the schema, or whether perhaps an "internal" and an "external" fibre tract connecting two centres merge into one for a long stretch. This would be totally irrelevant for the physiology of the speech function, though it would be highly important for the pathology of the cortical speech area. If Lichtheim's

presentation was based on new anatomical findings any further opposition would be impossible and most of the observations to be presented here would be pointless. But there is an even weightier objection to Lichtheim's schema: whenever one attempts to fit an observed speech disorder into it, difficulties arise, because one finds the individual speech functions disturbed in various degrees, instead of one being completely lost and another having remained intact. Furthermore, the ease with which speech disorders that cannot be explained through one single interruption in the schema, can be attributed to combined lesions, opens the door wide to arbitrary explanations. But while these are shortcomings which more or less adhere to every attempt at schematizing, Lichtheim's schema fails to satisfy one important requirement: by its very nature it must claim to be complete and to be able to account for every form of speech disorder observed clinically. Lichtheim already knew of a common instance which he was unable to fit into his schema, i.e., the combination of motor aphasia and alexia which is too frequent to be attributed to the coincidental interruptions of two fibre tracts. In trying to explain this syndrome Lichtheim made the assumption that these were cases with complete loss of all functions of speech in which the most easily reversible disability, i.e., word deafness, had already subsided; at that stage, therefore, only two other main disabilities, i.e., motor aphasia and alexia had remained. But this explanation does not seem to hold; Kahler[1] some time later reported the case of a patient who after his recovery from a transient aphasia maintained that at a time when he could only bleat instead of speak, and when he was

[1] Kahler: *Casuistische Beitraege zur Lehre von der Aphasie* (Clinical observations concerning the theory of aphasia). Prager med. Wochensch. Nr. 16 und 17, 1885.

unable to read because the letters appeared blurred, his understanding had been quite unimpaired throughout. Such and similar observations may have caused Eisenlohr,[1] one of the soundest German neurologists, to concede to Lichtheim's schema no more than a "chiefly didactic" value.

II

All authors since Wernicke have, explicitly or implicitly, adopted the view that speech disorders observed clinically, if they have an anatomical basis at all, are caused by lesions of the speech centres or by disruption of the speech association tracts, and that one is therefore justified in differentiating centre aphasias from conduction aphasias. It seems worth while to examine the validity of this distinction more closely as it is intimately related to Wernicke's afore-mentioned important concept of the rôle of the centres in the cortex and of the localization of mental functions.

If one recognizes the distinction between a "speech centre" and a mere connecting pathway consisting of a tract of white fibres, one has to expect a much more serious disturbance of function to result from destruction of a centre than from interruption of a conducting tract. Wernicke's presentation seems to bear this out. The

[1] Eisenlohr: *Beiträge zur Lehre von der Aphasie* (Contributions to the theory of aphasia). Deutsche med. Wochensch., Nr. 36, 1889.

only characteristic of his conduction aphasia caused by interruption of the tract a-b (Fig. 1) was confusion in the use of words, while in spontaneous speech the vocabulary was preserved and understanding unimpaired. The resulting disability, therefore, appeared to be much slighter than that in motor and sensory aphasia caused by lesions of the centres themselves.

However, there is something peculiar about Wernicke's conduction aphasia. The disturbance of function attributed to it cannot be deduced from Wernicke's schema. Wernicke states that interruption of a-b causes paraphasia; but if we ask what ought to be the result of this interruption the answer would have to be as follows: via the tract a-b the ability of reproducing perceived word sounds has been learned; its function is that of repetition of words heard; its interruption ought to result in a loss of that ability while spontaneous speech and understanding ought to remain intact. Yet everybody will admit that such a dissociation of speech functions has never been observed nor is it ever likely to be observed. The faculty of repeating is never lost as long as speaking and understanding are intact. It is absent only if (1) there is no speech at all, or (2) hearing is impaired. I know of only one single instance in which the ability to speak spontaneously and to repeat words perceived were not both intact: there are patients with motor aphasia who occasionally can produce a curse or a complicated expression which does not belong to their "speech remnants",[1] yet they are unable to repeat on request what they have just said. But this is an entirely different situation: these patients are equally incapable of spontaneously repeating these isolated additions to

[1] The phenomena described by this term correspond to those called "recurrent utterances" by Hughlings Jackson (Transl.).

their reduced vocabularies. The fact that there is no isolated loss of repetition of spoken language, and that this ability invariably remains intact as long as spontaneous speech is retained, is going to play an important part in the conclusion that one and the same tract serves speaking and the repetition of spoken words.

We are justified in denying the existence of Wernicke's conduction aphasia because it has proved impossible to find a speech disorder with the characteristics postulated. It was located by Wernicke in the insula. Lesions of this area must therefore produce a different type of speech disorder. In fact, Bastian,[1] in his excellent presentation of aphasia, makes the definite statement that lesion of the insula causes a typical motor aphasia. Unfortunately, the problem of the insular aphasia, which would be so very important for our considerations, has so far not been clarified by clinical observation. Meynert,[2] de Boyer,[3] Wernicke himself and others maintain that the insula belongs to the speech area, while Bernard[4] and others, following Charcot, emphatically deny such a relation. Nothing decisive concerning this problem emerged from Naunyn's[5] survey. Although it seems highly probable that lesions of the insula cause speech disorder (not only because of anatomical contiguity to the so-called

[1] Bastian, Charlton: *On different kinds of aphasia.* Brit. Med. J., Oct. 29 and Nov. 5, 1887.
Idem: *Brain as an Organ of Mind.* London, 1880.

[2] Meynert: Oesterr. Zeitsch. f. prakt. Heilkunde XIII.

[3] de Boyer: *Etudes cliniques sur les lésions corticales* (Clinical studies of cortical lesions) Paris, 1879.

[4] Bernard: *De l'aphasie et de ses diverses formes* (On aphasia and its various forms) Paris, 1885.

[5] Naunyn: *Ueber die Lokalisation der Gehirnkrankheiten* (On the localization of the diseases of the brain) Verhandl. IV., Congr. f. innere Medicin zu Wiesbaden, 1887.

centres), it is nevertheless impossible to state whether the speech disorder is of a specific type and if so of what type.[1]

We propose to postpone discussion of the symptom of paraphasia (mistaken use of words), and also of the reasons which caused Wernicke to regard it as characteristic of an interruption between a and b. At this stage we only want to mention that the paraphasia observed in aphasic patients does not differ from the incorrect use and the distortion of words which the healthy person can observe in himself in states of fatigue or divided attention or under the influence of disturbing affects,—the kind of thing that frequently happens to our lecturers and causes the listener painful embarrassment. It is tempting to regard paraphasia in the widest sense as a purely functional symptom, a sign of reduced efficiency of the apparatus of speech associations. This does not exclude that they may occur in most typical form as organic focal symptoms. Allen Starr[2] is the only author of distinction who has taken the trouble of searching for the anatomical causes of paraphasia. He arrived at the conclusion that this symptom could be produced by lesions of a great variety of localization; he found it impossible to establish a consistent difference in the pathology of cases of sensory aphasia with or without paraphasia.

It could be objected that the above criticism of Wernicke's conduction aphasia is unjustified because it

[1] Bastian (*On different types of aphasia*, 1887) is inclined to regard the combination of aphasia with hemianaesthesia described first by Grasset as the result of anatomical contiguity of the connections between Broca's and Wernicke's areas passing through the insula, and the posterior (sensory) third of the posterior peduncle of the internal capsule.

[2] Allen Starr: *The pathology of sensory aphasia, with an analysis of fifty cases, in which Broca's centre was not diseased.* Brain, XII, 1889.

does not allow for the following possibility: inability to repeat spoken language need not be manifest because the words heard which cannot be directly conveyed to the motor centre might be repeated through the detour via "understanding", the connection A-B-M (Fig. 3) taking the place of the interrupted tract A-M which normally serves repetition. If this detour is really available conduction aphasia would have to be characterized as a condition in which understanding and spoken language are intact, as well as repetition of comprehensible words, but in which repetition of incomprehensible words, such as those of a foreign language, is abolished. Such a syndrome has not been observed, though it has so far not been looked for. It may occur occasionally. In admitting this possibility we have to stipulate another condition arising from a strict differentiation between speech centres and their association tract. The destruction of a centre naturally results in an irreplaceable loss of function: if, however, only a pathway is severed it ought to be possible to stimulate the intact centre via some undamaged fibre tract and to mobilize its stored memories. In searching for a case in which such a compensating mechanism could be found we came across an instance the discussion of which is of the highest importance for the whole theory of aphasia.

There are cases of loss of understanding (word deafness) without disturbance of spontaneous language. They are rare, but they do occur, and the development of the theory of aphasia might have taken a different course had Wernicke's first case of sensory aphasia been of that type. However, his patients showed, like most of those observed later, the picture of sensory aphasia with an impairment of spontaneous language which we propose to call paraphasia in accordance with Wernicke. Such a speech disorder could not, of course, be explained

from his schema according to which the kinaesthetic word impressions are intact, as well as the pathways connecting them with the concepts. It is impossible, therefore, to understand why the words produced should not be correct. Wernicke had no choice but to base his explanation of paraphasia on the assumption of a functional factor which could not appear in his schema. He pointed out that the tract a-b, or A-M (Fig. 3), was the one via which speaking had been learned. Later on speech was produced directly from the concepts, but the tract a-b still retained a certain importance for speaking; whenever spontaneous speech was produced this tract was innervated also, and thus it exercised a continuous control over the flow of kinaesthetic impressions. Wernicke supposed paraphasia to be caused by loss of this associated innervation.

Wernicke's ideas about this difficult problem are far from clear and, I believe, not even consistent, because in a later passage (p. 23, l. c.) he expressed the view that the mere existence of the tract a-b without its innervation was sufficient to guarantee the choice of the correct kinaesthetic impression. Wernicke's presentation has not enabled me to form a clear and unequivocal picture of how the mere existence of the fibre tract, even without associated innervation, can possibly have such a powerful effect on the motor speech process; nor how a collateral innervation, if it takes place at all, can manifest itself; nor whether the centre b transmits the impulse to articulation only when the stimulus from centre a has been received; nor whether it starts functioning earlier, makes mistakes and corrects them through the excitation of the word sound centre. Lichtheim must have been aware of Wernicke's failure to explain the symptom of paraphasia, because he defined the conditions under which paraphasia did not occur much more concisely. In his view

the auditory word images had to be intact, as well as their connection with the kinaesthetic word impressions through the tract a-b. Had Lichtheim gone a step further he would have had to assume that speech was produced only via the sound images and the tract A-M, because any controlling influence of A over the production of speech via A-M is entirely useless if it becomes effective only after words have been uttered from M; speech is obviously not produced before this impulse has been received in M, and all difficulties dissolve if we drop the superfluous assumption that a special stimulus from the "concepts" to M is necessary for speech.

However this may be, according to Wernicke and Lichtheim spontaneous speech in sensory aphasia (destruction of A) becomes paraphasic because the sound images in A which normally have a controlling function have been destroyed. One would expect the clinical picture to be different if these important sound images remain intact and only the fibre tract connecting them with B has been destroyed. Such a difference would convince us that lesions of fibre tracts have a different significance from those of centres, and that images are contained only in the latter and not in the former. We should have to assume that the intact sound images exert their influence on speaking through the detour via the "concept centres" in a way similar to that contemplated earlier when repetition was discussed. In Wernicke's conduction aphasia the centre is intact, but the association fibres are interrupted; and yet such a detour is not made. The interruption of A-M has the same effect as the destruction of A itself, i.e., paraphasia in spontaneous speech. This is another proof that Wernicke's conduction aphasia is untenable. If we assume that interruption of a-b (A-M) cannot be compensated for by some detour of innervation, inability to repeat

would be inevitable. If, however, we allow for the possibility of such a detour paraphasia ought not to occur. Consideration of all the conduction aphasias postulated by Lichtheim, and of the disorders of reading and writing not caused by lesions of the centres, leads to the following conclusions: the destruction of a so-called centre comes about only through simultaneous interruption of several fibre tracts; any assumption of a centre lesion can be replaced by one of a lesion of several tracts, without abandonment of the theory of localization of psychic functions in the areas of the centres.

As I feel rather isolated in claiming that the alleged psychological status of the speech centre ought to manifest itself in the symptoms of the speech disorders in some way, I hasten to refer to a short but significant paper by de Watteville[1] who expressed a very similar line of thought. "We have formed the idea", he wrote, "that these centres are storage places in which the various motor and sensory memory images are preserved. On the other hand, we must not search for the physiological substratum of mental activity in this or that part of the brain but we have to regard it as the outcome of processes spread widely over the brain. It follows from these two premises that certain lesions, the gross symptoms of which do not differ materially, must still differ in their psychological effects; let us take two cases of motor aphasia, one of them caused by destruction of Broca's centre itself, the other by interruption of the centrifugal tract originating from it. In the first case, the patient has lost control over his kinaesthetic word impressions, in the second this control has remained unimpaired. The effect of aphasia on intelligence and vice versa has been discussed frequently, yet in spite of

[1] de Watteville: *Note sur la cécité verbale* (Note on word blindness). Progrès médical. March 21, 1885.

good observations on both aspects the results have been full of contradictions. Might this not be due to the conditions mentioned above? . . . We feel justified in assuming that if aphasia has been caused by a central lesion the patient must have suffered intellectual damage also, while this need not be the case if the fibre tracts only have been damaged."

I do not think that anybody has taken the trouble to carry out the examinations necessary to prove Watteville's hypothesis; I doubt that a more severe intellectual impairment will be found to be associated with "central" aphasia than with a conduction aphasia.

III

We have endeavoured to establish which clinical features bear out the alleged psychological significance of the speech centres and have for this purpose subjected Wernicke's conduction aphasia to a critical examination. In doing so we discovered certain facts which gave rise to serious doubts in the fundamental correctness of a schema based mainly on localization, such as that of Wernicke and Lichtheim. It should not be overlooked, however, that both authors also invoke, without hesitation, functional factors in the explanation of speech disorders. A presentation which attempted to explain the variety of speech disorders observed by differences in the localization of the lesions only, would have to confine

itself to assuming a number of centres and tracts functioning independently and equally liable to be put out of action. Wernicke and Lichtheim had to concede that the function of the motor centre M depended not only on its anatomical integrity but also on the maintenance of its connection with the sensory centre A. Indeed, Lichtheim made a surprising observation, the confirmation of which would reduce the significance of localization even further. He posed the question whether motor aphasics were in possession of "inner language", i.e., whether they could recall the sound of words which they were unable to express. He asked them to squeeze his hand once for each syllable of the requested word, and found that they were unable to prove their knowledge of the word in this way. This observation is bound to have a profound influence on the conception of the speech process for the following reason: the centre A is intact and its connection with the rest of the cortex unimpaired; the only part damaged is M, the centre of the kinaesthetic word impressions; and yet the patient is unable, because of a circumscribed lesion in the third frontal convolution, to elicit the word sound contained in the temporal lobe even with the help of some other cerebral activity such as the visual perceptions.

Unfortunately, this observation, which ought to be the corner stone of a new theory of the aphasias, has so far not been established beyond doubt. First of all, there are some objections to the way in which Lichtheim set out to prove it. His criterion of the availability of the word sound was the patient's ability to state the number of syllables of the word wanted; but it can be assumed that these patients had been in the habit of arriving at that number by transferring the sound to the motor speech tract. In this case the test would have been

unsuitable because it implied the integrity of the very tract that is destroyed in motor aphasia. A similar objection against the validity of Lichtheim's test has been raised by Wysman.[1] But there is still another objection: Lichtheim reported that he had been unable to apply his test in cases of typical cortical motor aphasia (with destruction of M) because he had had no pure cases of that type at his disposal for some time. He reported only a case of so-called transcortical motor aphasia in which the test was negative, although in this type of disorder not even M is supposed to be damaged but only its connections with B. However, I shall later demonstrate that these cases of transcortical motor aphasia call for a different hypothesis which is compatible with the loss of the sound images. The question whether or not the sound images are available in motor aphasia still appears to be undecided. Yet I should hesitate to advance a theory of aphasia before I had definite knowledge on this point.

Let us now return to the two other arguments on which we base our rejection of the functional independence of the centre M. (1) If there was a connection between M and B (tract for spontaneous speech) which was different from the connection between M and A (the tract which makes repetition of words heard and correct speaking possible), we ought to find disturbances of repetition without corresponding impairment of spontaneous speech. We have established beyond doubt that this does not happen. We therefore conclude that these two tracts are in fact one and the same.

(2) A lesion in A or in the tract A-M causes a speech disorder which compelled Wernicke and Lichtheim to adduce functional factors, without, however, enabling

[1] Wysman: *Aphasie und verwandte Zustände* (Aphasia and kindred conditions). Deutsch. Arch. f. klin. Med. Vol. 47.

them to explain satisfactorily the occurrence of paraphasia in sensory aphasia. This difficulty, also, is resolved if one assumes that there is only the tract A-M, and that spontaneous speech takes place only through sound images (sound impressions). This assumption is all the more suggestive as A-M is undoubtedly the first tract via which the child learns to speak. Wernicke assumed that after speaking via this tract had been sufficiently practised, another more direct tract which had no connection with the sound images came into use. But it is impossible to understand how practice in the use of a fibre system should result in its abandonment and in the choice of another. Almost all earlier writers, including Kussmaul,[1] insisted that spontaneous speech took place via the same pathway as repetition, i.e., by means of the sound images, and a more recent author, Grashey,[2] has reverted to this assumption. I have never been able to understand the arguments with which Lichtheim in his otherwise lucid presentation defends his thesis of a direct motor speech tract against Kussmaul.

If we suppose the pathway for spontaneous speech to go via the sensory centre A, the speech disorder resulting from a lesion of that centre naturally assumes particular interest for us. Indeed we have the impression that Wernicke and Lichtheim have not done full justice to it by calling it "paraphasia". By paraphasia we are to understand a speech disorder in which the appropriate word is replaced by a less appropriate one, which, however, still retains a certain relationship to the correct word. Following the

[1] Kussmaul: *Die Störungen der Sprache* (Disorders of speech). 1877.

[2] Grashey: *Ueber Aphasie und ihre Beziehungen zur Wahrnehmung* (On aphasia and its relationship to perception). Arch. f. Psychiatrie, Vol. 16, 1885.

philosopher Delbrueck[1] we may describe these relations as follows: In paraphasia words of a similar content, or linked by frequent association, are used in place of one another, e.g., "pen" instead of "pencil", "Potsdam" instead of "Berlin". Furthermore, words of a similar sound are mistakenly used for each other, such as "Butter" for "Mutter" or "Campher" for "Pamphlet"; and finally, if the patient makes mistakes in articulation (literal paraphasia), single letters are replaced by others. It is tempting to differentiate between various types of paraphasia according to the part of the speech apparatus at which the mistake took place.

One also speaks of paraphasia when two intended words are fused into one malformation, such as "Vutter" for "Mutter" or "Vater"; by common consent circumlocutions by which a specific noun is replaced by a very general one ("dings", "machine", "chose") or by a verb, have also been regarded as paraphasia. However, the speech disorder of sensory aphasia may go far beyond paraphasia. There are aphasics who do not produce any comprehensible words, but pour forth an endless sequence of senseless syllables (gibberish, jargon aphasia of English authors).[2] In other cases, poverty of words of any specific meaning, abundance of particles, interjections and other grammatical accessories, and frequent repetition of nouns and verbs are conspicuous. One of Wernicke's patients, whose aphasia had already improved considerably, produced the following sentences in response to a present given to her: "*Da lass ich mir viel viel Mal alles Mögliche, was Sie nur haben gesehen. Ich danke halt viel liebes Mal, dass Sie mir das alles gesagt. Na, da*

[1] Delbrueck: *Amnestische Aphasie* (Amnesic aphasia). Jena'sche Zeitsch. f. Naturwissensch. XX. Suppl. II., 1886.

[2] Ross: *On Aphasia.* London, 1887 (also Manchester Medical Chronicle).

danke ich vielmal, dass Sie mir das alles gesagt. Na, da danke ich vielmal, dass Sie sind so gut gewesen, dass Sie sind so gütig gewesen." ("There I leave for myself many many times everything possible which have you only seen. I thank many a good time that you told me all this. There I thank many times that you have been so kind, that you have been so kindly.") I remember having myself seen a case of sensory aphasia in the Vienna General Hospital, a Mrs. E. who was demonstrated to us as "encephalitic confusion"; her speech showed the same peculiarities: impoverishment in nouns, adjectives and verbs; abundance of all other types of words, and a tendency to reiteration. Wernicke regarded "an intact vocabulary with paraphasia" as characteristic of the sensory aphasia. I believe it can be more correctly described as "impoverishment of words with an abundance of speech impulses".

However, if we omit the tract for spontaneous speech, B-M, from Lichtheim's schema, how shall we explain the cases of transcortical motor aphasia which Lichtheim so easily explained with the interruption of that very pathway? These are the cases in which spontaneous speech is quite impossible, while repetition, reading aloud (i.e., speaking from visual images), etc., proceed unimpaired.

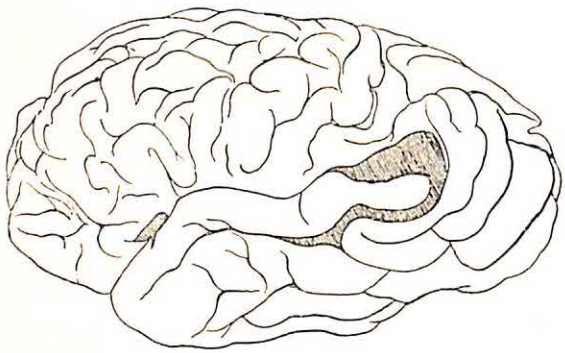
Fortunately we can arrive at an understanding of these cases in a different way. Heubner[1] recently published an important observation to which we shall frequently refer. His patient had lost the capacity of speaking spontaneously, but he had retained the ability to repeat words spoken to him by others, and to read aloud. He also had lost comprehension of spoken and written language. The symptoms were those of a combination

[1] Heubner: *Ueber Aphasie* (On aphasia) Schmidt's Jahrbücher, 1889, Vol. 224, p. 220.

of a transcortical motor with a transcortical sensory aphasia. His case could not be explained by entering a single lesion in Lichtheim's schema, but rather by the coincidence of two lesions: one in the tract B-M and another in the tract B-A. However, the post mortem examination revealed a cortical softening of a most interesting localization, at any rate as far as the sensory speech area was concerned; it encircled Wernicke's area in the first temporal convolution, thus isolating it from the rest of the cortex on its upper posterior and lower circumference. There was, in addition, a superficial cortical softening, the size of a lentil, on the tip of a gyrus belonging to the third frontal convolution (Fig. 5).

These findings seemed at first to corroborate Lichtheim's schema, but on closer consideration one

FIG. 5



The post mortem finding in Heubner's case.

must agree with Heubner that the lesion in the motor speech area was much too limited and insignificant to be charged with the "enormous and profound disturbance of speech". Besides, it was situated in the cortex and was in no way transcortical, and if it had caused disturbances they would have interfered with repetition as well as with spontaneous speech. The speech disorder has to be

explained by the considerable lesion in the sensory area only, and we see from this case that isolation of the sensory centres from its cerebral connections, i.e., a transcortical sensory lesion, can also cause loss of spontaneous speech. This means that the tract B–M is identical with the tract B–A, or that speech is produced only via the sound images.

We remember that Lichtheim, with the aid of his syllable test, established in his case of subcortical motor aphasia that the patient was unable to elicit sound images with the help of his "concepts". If we may draw inferences from Heubner's case to that of Lichtheim in which the speech functions were less severely disturbed, we may assume that in the former case the lesion was probably also situated in the sensory area. If this should be correct the negative result of Lichtheim's test would lose the significance it would have had if the lesion in the motor speech area had been verified.

It is admittedly unsatisfactory to base a decision on one single case, all the more so as there was a small lesion in the motor area. I have therefore endeavoured to find some other cases of so-called transcortical motor aphasia with post mortem findings, and I have arrived at the following unexpected conclusion: loss of spontaneous speech which is not coupled with inability to repeat words perceived, does not by itself indicate a lesion of the sensory area. This symptom, which is characteristic of the transcortical motor aphasia, can also be found in cases with a lesion confined to the motor area alone; but in one case only could the lesion be correctly described as transcortical. In this case (Magnan[1]) a tumour was found which had originated from the inner surface of the dura. It had proliferated

[1] Magnan: *On simple aphasia and aphasia with incoherence.* Brain, II, 1880.

into the left hemisphere like a wedge the thin edge of which had reached the third left frontal convolution and the anterior third of the upper margin of the insula. The patient was unable to give information about herself, being able to utter only single words and senseless syllables; but she could repeat words spoken to her.

In the two other cases in which post mortem findings were available the lesions were situated in the motor cortex itself. They could be called "transcortical" only if one used this term in a sense which makes it quite useless in the theory of aphasia. In one case the lesion consisted of a haemorrhage in the motor centre, in the other it had been caused by a bone fragment lodged within that centre. Both cases were observed by Hammond[1] who described them as follows:

Case I. When Hammond, in summer 1857, was stationed in the Rocky Mountains with a group of soldiers and labourers, a Mexican was hit by a workmate on the left temple with a club and collapsed unconscious. When he recovered consciousness he had completely lost his memory for words but not the ability to articulate. He was incapable of spontaneous speech but able to repeat words spoken to him with correct articulation provided they were only a few at a time; e.g., when Hammond asked him "*come sientes ahora?*" (How are you now?) he repeated, "*Come sien, sien, sien*", and then burst into tears. The patient died on the following day; the post mortem examination revealed a "haemorrhage of the size of a half dollar piece involving the left frontal lobe on its postero-lateral margin" and a rupture of the right middle meningeal artery.

Hammond's clinical examination of this case can

[1] Hammond: *A Treatise on the Diseases of the Nervous System.* Seventh edition. London, 1882.

hardly have been exhaustive; he added to his report: "I did not attribute any special importance to the lesion of the left frontal convolution at the time. Only after the discussion in the Paris Academy in 1861 did I realize that this patient's amnesic aphasia had been caused by the lesion."

Case II. During the winter of 1868-69 Hammond saw a man who some months previously, when working in a quarry, had sustained a knock against the left side of his head from a machine. The patient appeared intelligent, understood everything that was said to him, and made the most desperate effort to express himself; but he was unable to utter any words except "yes" and "no". Hammond asked him: "Were you born in Prussia?"—"No"—"In Bavaria?"—"No"—"In Austria?"—"No"—"In Switzerland?"—"Yes, yes, yes, Switzerland, Switzerland." When giving the last answer he laughed and gesticulated wildly. Hammond assumed that the accident had caused a fracture of the inner table of the skull and that a bone fragment was pressing on the third frontal convolution. On his advice trephining was carried out and his diagnosis was confirmed. As soon as the patient woke up from the anaesthesia his speech was completely restored.[1]

In these cases Lichtheim's transcortical motor aphasia had been caused by lesions which had nothing whatever to do with interruption of a tract B-M.

On closer consideration of these cases another important aspect emerges which might be relevant for other speech disorders also. It is generally known that in the great majority of cases motor aphasia is caused by softening. It seems a remarkable coincidence that in

[1] Hammond's report of these two cases is not fuller than the one given here. Lichtheim, nevertheless, classified the first case as one of transcortical motor aphasia. I beg to venture the same opinion concerning the second case.

each of the above-mentioned cases of so-called transcortical motor aphasia the lesions were of a different nature, except for Heubner's case which showed a sensory disturbance. Lichtheim's principal case was traumatic, and so were Hammond's two cases. Finally, in Magnan's case the speech disorder was due to a tumour.[1]

If lesions of the brain give rise to symptoms at all, conclusions as to the localization of the damage can be drawn, whereas we have to guess the diagnosis of the pathological process from special circumstances of the case or from the course of the illness. The speech apparatus, however, is exceptional, in having at its disposal such a wealth of symptoms that it may be expected to betray, by the type and manner of the disturbance of function, not only the site but also the nature of the lesion. Perhaps one day we shall be able to differentiate clinically aphasias due to haemorrhage from aphasias due to softening, and to recognize certain speech disorders as characteristic of specific pathological processes affecting the apparatus of speech.

It can be regarded as established that the occurrence of the so-called transcortical motor aphasia at any rate, does not prove the existence of a special pathway B-M for spontaneous speech. This type of speech disorder is caused either by lesions in the sensory speech area or by special affections of the motor speech region as the result

[1] In the case of transcortical motor aphasia referred to by Lichtheim (case of Farge, quoted by Kussmaul, p. 49, and in Nothnagel's *Topische Diagnostik*, p. 358), a softening in the white matter adjoining the third left frontal convolution was found. Nothnagel denied that this case by itself proved anything about the significance of subcortical lesions for the origin of aphasia as the patient had died twenty days after the softening had set in; at that time, he argued, remote effects of the lesion on the third frontal convolution, which may appear anatomically intact, could not be ruled out.

of which the motor centre is functioning at a lower level.[1]

Charlton Bastian[2] with whose explanation of the so-called transcortical motor aphasia we agree, distinguishes three states of reduced excitability of a centre. The smallest reduction manifests itself in a failure of the centre to react to "volitional" stimulation while it still reacts to stimulation by association with another centre and to direct sensory stimuli. If function is more severely disturbed the centre reacts to direct sensory stimulation only, and finally, at the lowest level of functioning, that reaction also fails. For the transcortical motor aphasia one would, therefore, have to assume that the motor centre can still be activated by direct sensory stimulation while volition no longer has this effect; and as this motor centre is always stimulated by association with the auditory centre, the cause of the change of excitability may be situated in either.

Our considerations have led us to attribute a certain clinical type of speech disorder to a change in the functional state of the speech apparatus rather than a localized interruption of a pathway. As this step is so very important for the whole theory of aphasia, we propose, in order to make sure of our premises, to restate that we were compelled to drop the localizatory explanation because the post-mortem findings (Heubner, Hammond) had failed to bear it out. The assumption

[1] The following is a list of the six cases of transcortical motor aphasia in which the etiology was established. (1) Lichtheim: traumatic contusion of unknown localization. (2) Farge: indirect interference with the motor speech area by a softening in its vicinity. (3) Heubner: softening in Wernicke's area. (4) Magnan: tumour involving Broca's area. (5) Hammond I: traumatic haemorrhage over Broca's area. (6) Hammond II: inhibition of the motor speech area through a bone fragment lodging in it.

[2] Charlton Bastian: *On different kinds of aphasia.* Brit. Med. Journ., Oct. 29 and Nov. 5, 1887.

made by Bastian and ourselves appears to follow without difficulty from the fact that repetition invariably remains intact longer than spontaneous speech. Later on we shall produce observations which will also demonstrate that the associative activity of a centre is less easily lost than the so-called spontaneous one.

Bastian's hypothesis seems at first somewhat perplexing; it appears irreconcilable with an approach aimed at the study of localized lesions and their effects. It may be argued that reduction of excitability of a centre, being a purely "functional" state, does not imply the presence of an actual lesion. This is correct, and there might be conditions similar to the transcortical motor aphasia which are the result of mere functional impairment without organic damage. However, if one considers the relationship between "organic lesion" and "functional disturbance" one must realize that a great number of organic lesions cannot manifest themselves otherwise than by disturbances of function, and experience shows that these lesions have, indeed, no other effect. For decades we have been endeavouring to advance our knowledge of the localization of functions by the study of clinical symptoms; we have got into the habit of expecting a lesion to destroy a number of units of the nervous system completely and to leave the rest completely intact, because only thus, we believe, can clinical experience be made to fit our preconceptions. Yet only few lesions comply with these postulates. Most lesions are not directly destructive and they have a disturbing effect on much larger a number of nervous units than those immediately involved.

Furthermore, the impact of an only partly destructive lesion on the whole of the apparatus concerned must be taken into consideration. Two possibilities are conceivable both of which do in fact occur. Either some parts

of the apparatus are put out of action by the lesion while the intact parts continue to function as usual; or it reacts to the lesion as an unitary whole, in which case there is no loss of part functions but a lowering of function in general. To an incomplete lesion it responds by a disturbance of function which could be caused also by non-structural damage. The central motor apparatus for the upper extremities, for instance, shows both modes of reaction: a small lesion in the anterior central gyrus may cause an isolated paralysis of the muscles of the thumb. More commonly, however, it results in a slight paresis of the whole arm. It appears that the speech apparatus shows in all its parts the latter kind of reaction to incomplete lesions; it responds to such a lesion with a disturbance of function. For instance, a small lesion in the motor speech area would never cause the loss of a hundred words the type of which would depend only on the site of the lesion. Partial loss can always be shown to be the expression of a general lowering of the functional activity of that centre. It is not, by the way, a matter of course that the speech centres should behave in this way; their reactions to damage suggest a certain concept regarding their organization which is to be discussed later.

Before leaving the subject of motor aphasia it seems appropriate to consider two points: if transcortical motor aphasia is symptomatic of a state between normality and complete loss of excitability one would have to expect it to occur when motor aphasia is in the process of subsiding, i.e., one would anticipate motor aphasics to pass through a phase when they are better able to repeat words heard than to speak spontaneously. I believe that a case described by Ogle[1] bears this out. I have not been

[1] Quoted by Bastian: *On the various forms of loss of speech in cerebral disease.* Brit. and Foreign Med.-Chir. Review, Jan. 1869.

able to find other instances which would confirm my expectation. I daresay that clinicians have not directed their attention to this question.

Secondly, I have to consider an objection which has no doubt occurred to every reader: if spontaneous speech takes place via the sound images by the route B-A-M, every sensory aphasia ought to result in loss, and not only in a disturbance, of speech. How is it to be explained that, on the contrary, in this type of aphasia speech is still so abundant though incorrect? I can do no more than recognize the difficulty and point to another one in reply.

There are cases of logoplegia, i.e., of simultaneous loss of understanding and of expression, where our postulate of the loss of spontaneous speech in sensory aphasia seems to be met. However, in these cases the disability is caused by multiple and extensive lesions involving both motor and sensory areas. These cases seem to take a characteristic clinical course: the sensory disorder subsides and in a later stage the patient presents the picture of a pure motor aphasia. It may also happen that the patient presents a motor aphasia from the beginning, and post-mortem examination may reveal a lesion affecting not only Broca's region but also a large part of the remaining speech region including Wernicke's area. Kahler[1] has reported a case of this type which is far from rare, and has collected similar cases from the literature. The occurrence of cases with lesions of the sensory centre but without word deafness, at least without permanent word deafness, has been established beyond doubt, although every word deafness is to be related to a lesion of that centre. How this apparent contradiction

[1] Kahler: *Casuistische Beiträge zur Lehre von der Aphasie* (Clinical observations concerning the theory of aphasia). Prager med. Wochensch., Nr. 16 and 17, 1885.

can be resolved I am at present unable to state; but I believe that its clarification will also provide the answer to the earlier question why sensory aphasia does not always entail a complete loss of speech. It is important to realize that the extension of the centre A has not been finally established.

There are cases of sensory aphasia without any disturbance of spontaneous speech, cases with only slight paraphasia and marked impoverishment of language and cases with distortion of speech amounting to gibberish. According to Allen Starr,[1] it is impossible to explain these variations by differences in the localization of the lesions within the sensory area. Perhaps some observations to be presented later in this book will contribute to the clarification of this difficulty.

IV

The publication of Lichtheim's paper which presented the localizatory theory of aphasia with such consistency, coincided with an address by Grashey[2] which was soon hailed as a fundamental contribution to the understanding of aphasia, although hardly anybody has

[1] Allen Starr: *The pathology of sensory aphasia, with an analysis of fifty cases in which Broca's centre was not diseased.* Brain, XII, 1889.

[2] Grashey: *Ueber Aphasie und ihre Beziehungen zur Wahrnehmung* (On aphasia and its relationship to perception). Archiv f. Psychiatrie, XVI, 1885.

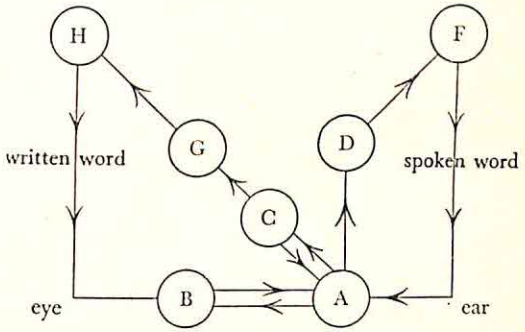
continued on his lines. Grashey's case showed no special features, except one; the patient was a man aged 27, who as the result of a fall had sustained a fracture of the skull; he was almost completely deaf in the left ear, had lost his sense of smell and taste, was able to see with his right eye hand movements only, while the visual acuity of his left eye was reduced to 2/3 and his visual field was contracted. The facialis and hypoglossus, as well as the whole musculature of the right side of the body were paretic. In addition, the patient had a speech disorder which immediately after the injury showed the features of word deafness. When he came under Grashey's observation his speech had greatly improved and showed only some of the common residual disturbances. The patient was able to speak coherently, he used all prepositions and conjunctions and also some verbs and adjectives without difficulty; in spontaneous speech he produced an occasional noun, but usually resorted to circumlocutions ("Dingsda"). He recognized every object which he had known before his illness yet was unable to name any. Understanding of spoken language was intact. The inability to use nouns in spontaneous speech and to name objects, though they were recognized, is known to be one of the most common symptoms of the so-called amnesic aphasia which was distinguished from the atactic aphasia by earlier writers.[1]

The relationship of amnesic aphasia to the types of speech disorder which can be attributed to interruption of pathways has always been a difficult problem. This is not surprising as the concept of amnesic aphasia was based on psychological, and that of the other forms on anatomical considerations. Lichtheim regarded it as incorrect to list amnesic aphasia together with the other

[1] The distinction between amnesic and atactic aphasia was proposed by Sanders in 1866.

speech disorders. In his view it was a common accompaniment of the typical aphasias and their residual states; it was not a focal symptom and occurred in cases with more diffuse pathological processes, such as

FIG. 6



The schema with the help of which Grashey explained the disturbance of function observed in his patient.
A centre for sound images; *B* centre for object images; *C* centre for symbols, i.e., written and printed letters, words and numerals; *D* centre for the kinaesthetic impressions of articulate speech; *F* nuclei of the nerves serving phonation and articulation; *G* centres for the kinaesthetic impressions of writing movements; *H* nuclei of the motor nerves serving writing movements.

generalized vascular lesions, or as a sign of senile reduction of cerebral functions.

The suggestion that the principles of localization which had been declared all-important for one class of speech disorders, should not be applied to another class, seems at first quite unconvincing. Grashey undertook an analysis of the symptoms of his case of amnesic aphasia with the help of the schema reproduced in Fig. 6. He considered the possibility that the pathway from the area of the sound images to that of the object images was intact, while the pathway in the opposite direction was interrupted. Under these circumstances the patient might have been capable of correctly relating a word to the appropriate object, yet unable to find the sound

impression for the object presented to him. Grashey, very much to his credit, dismissed this hypothesis with the following words: "After all, in this way one could explain any symptom. . . . I was therefore not satisfied with the arbitrary insertion and elimination of conducting tracts, but examined the patient more carefully. I found that the functions of the apparently normal centres were considerably disturbed." His patient showed a conspicuous inability to retain "object images, sound images and symbols" over any length of time. When shown an object which he was able to recognize, and requested after a few moments to touch it, he had in the meantime forgotten which object he had been shown; when two words were said to him in succession he was unable to repeat the first when requested to do so; he had invariably forgotten it and had retained the second word only. For the same reason, he was unable to synthetize, and to perceive as wholes "object images, sound images, impressions of touch, and symbols". If the picture of an object known to him was covered by a piece of paper with a slit in the middle, and if this paper was moved about so that only successive parts of the picture became visible, he was unable to put the visual impressions of these parts together; yet when the paper was removed he could at once see and recognize the picture as a whole. When a written or printed word was covered in the same way, letters being exposed singly in succession, he read them one by one but was unable to read the word; having arrived at the last letter he had forgotten the preceding ones.

Grashey explained his patient's speech disorder by this general impairment of perception, without postulating a localized lesion. An object, he argued, could be perceived visually even if exposed only for a brief fraction of time. A sound image he believed to require a longer period to

be perceived because it needed time to develop from successive stimuli. If the time allowed for the visual perception of an object was as low as 0.06 sec., it could still be perceived as a whole, while of its respective sound impression, i.e., its name, only the first letter could be perceived auditorily in that space of time. However, the parts of the object images and of the sound images did not correspond; the sound of the word "horse" had no relation to any part of the object "horse"; the sound image had to be complete before it could be related to the object. "If therefore, an object image is to elicit a sound image, the former must be complete and continue long enough to allow the successive parts of the sound image to emerge. If the time allowed for exposition of the object image 'horse' drops below 0.06 sec., only a fraction, i.e., a single letter of the sound image can be elicited."—"If, on the other hand, an image of the object is to be elicited from the sound image no part of the emerging sound image can elicit a part of the visual object image because the parts of the images do not correspond to each other. The sound image must be complete and continue long enough to enable the image of the object to emerge." As the object image required only a moment to emerge a sound image of very short duration sufficed.

Grashey concluded that one and the same disorder was apt to alter the transition from images of objects to the sound images, while the transition of sound images into object images remained unchanged. We add: without the assumption of a lesion in any tract or centre.

Grashey's patient showed yet another peculiarity. He was able to recall the names of objects by writing them, but only when allowed to look at the object at the same time. He would glance at it and write down the first letter of its name, read that letter and repeat it several

times; then he would look at the object again, write the second letter, pronounce the first two letters and continue in this manner until he had produced the last letter and with it the required name. This peculiar procedure could be satisfactorily explained with an abnormally short duration of the single perceptions if one presumed that the writing down and reading of the letters were means of fixating the fleeting perceptions. Grashey was justified in concluding from this observation that successive parts of the sound images, of the cheiro-kinaesthetic and the visual word images corresponded to each other, and that their association could help in eliciting the word even when the duration of the single perception had declined considerably.

It therefore appeared proven that there were cases of aphasia in which no localized lesion needed to be assumed and the symptoms of which could be attributed to an alteration of a physiological constant in the speech apparatus. "Grashey's aphasia" could be clearly distinguished from the aphasias described by Wernicke and Lichtheim and caused by localized lesions. It seemed possible that discovery of further functional mechanisms other than the reduction of the time of exposition of sensory impressions may result in clarification of other forms of "amnesic aphasia".

However, Wernicke[1] himself subjected Grashey's analysis to a trenchant criticism and demolished its basic assumptions. He pointed out that the sound image was not perceived as a sequence of letters. The word sound was a whole, which only later in life could be broken down into sounds of letters to meet the requirements of writing. Nor did Wernicke overlook another weighty objection to Grashey's hypothesis: if the patient had

[1] Wernicke: *Die neueren Arbeiten über Aphasie* (Recent studies of aphasia). Fortschr. d. Medicin, 1885, p. 824; 1886, pp. 371, 463.

built up the word sound from the sounds of the constituting letters, his hearing could not have been better than his reading, and he would have been unable to understand one single word without putting it down in writing. Wernicke expressed this objection thus: "The same patient who, when shown various successive objects or letters, invariably forgets the preceding ones, can read fluently, understands everything said to him and can write to dictation. To understand a word or sentence, the sound of several successive letters and words respectively must be retained by the patient long enough to enable their meaning to be comprehended. The sound images, therefore, are in this case retained much longer than visual object images and the memory disorder is in a certain sense localized, i.e., it concerns chiefly the area of visual activity." (p. 470.)

We note that Wernicke was unable to explain Grashey's case without assuming a localized and select functional disturbance. However, the emphasis on the visual dysfunction cannot satisfactorily explain the peculiarities of Grashey's case. We also remember that Grashey established that the sound images, too, were of extraordinarily brief duration in his case. Furthermore, unless the duration of the sound images had been markedly reduced, it would be impossible to understand why the patient needed to fixate them, by writing and reading, once they had emerged; he ought to have arrived at the whole of the sound image without special help if the perception of the object was renewed often enough.

Grashey's case therefore, calls for a different explanation and I hope that the one to be presented here will prove unassailable. The general reduction in the duration of sensory impression obviously cannot result in a speech disorder such as the one under discussion.

Rieger[1] carefully examined a patient with a very similar memory disorder, also caused by a trauma, and he paid due attention to the patient's speech. He had difficulties in finding nouns and adjectives in spontaneous speech and required constant coaxing to produce the names of objects shown to him. He succeeded in doing so only after a long interval. This was not used for building up words by way of spelling, but they were uttered explosively. All this suggests that in Grashey's case a localized lesion must have been present in addition to the general impairment of memory, and that the lesion was situated in the centre for the sound images. The case presented an example of Bastian's second level of reduced excitability when a centre fails to respond to normal, i.e., volitional, stimulation, but is still reacting to association and sensory stimulation. In Grashey's case the centre for the sound images could no longer be stimulated directly through object associations, but still permitted the conduction of the stimuli to the visual word images associated with the sound image. Of the former, the first part (letter) could be recognized during the fraction of time when the stimulus arising from the seen object took effect; through repetition of this process the other parts emerged. The letters of the visual word image thus assembled elicited the sound image which could not be activated through the object associations alone.

My interpretation is strongly supported by the fact that Grashey's patient was at first word deaf, which implies the presence of a gross damage in the very area a moderate lesion of which would explain the speech disorder described by Grashey. I am, of course, still of

[1] Rieger: *Beschreibung einer Intelligenzstörung infolge einer Hirnverletzung nebst einem Entwurf zu einer allgemein anwendbaren Methode der Intelligenzprüfung* (Description of a disorder of intelligence due to a brain injury, together with a design for a generally applicable method of testing intelligence). Wiesbaden, 1888.

the opinion that the auditory part of the speech apparatus reacted as a whole to this lesion, in the same way as it did in the transcortical motor aphasia discussed earlier.

Cases such as that described by Grashey have been known before. A patient reported by Graves[1] had, following a stroke, lost his memory for nouns and adjectives, but was able to remember their first letters with unfailing certainty. He found it helpful to draw up an alphabetic list of the most commonly used nouns which he always carried with him. When he required a word he looked it up under its first letter; having recognized the wanted noun by its visual word image he was able to pronounce it as long as he kept his eyes fixed on the written word. As soon as the book was closed he forgot the word. It is obvious that this patient, too, could make missing words available by establishing an association with their visual word images.

In the study of speech disorders it has often been observed that, for speech to be produced, the activity of a centre requires to be assisted by the activity of another associated with it. The visual centre (area of the letter images) shows this need most frequently, and in such cases reading is impossible unless the individual letters are copied, or drawn in the air. Westfal was the first to report such an observation in an aphasic patient who could read only when carrying out writing movements at the same time. In Charcot's[2] *New Lectures* which I have recently translated, we find the full history of another

[1] Quoted by Bateman: *On aphasia or loss of speech* etc., London, 1870.

[2] Charcot: *Neue Vorlesungen über die Krankheiten des Nervensystems, insbesondere über Hysterie* (New lectures on the diseases of the nervous system, especially on hysteria). Translated by Sigm. Freud. Wien, 1886, p. 137.

word blind patient who availed himself of the same mechanism. Thus the aphasias simply reproduce a state which existed in the course of the normal process of learning to speak. As long as we were still unable to read fluently, we endeavoured to make sure of the visual word images by arousing all other images associated with them, and equally, when learning to write, we used to stimulate the sound images and the motor kinaesthetic images, in addition to the visual word images. There is only one difference; when learning, we are restricted by the hierarchy of the centres which started functioning at different times; the sensory-auditory first, then the motor, later the visual and lastly the graphic. In pathological cases, however, the centre which has suffered least is the one the assistance of which is sought first. The cases of Graves and Grashey were peculiar only in that the centre of the sound impressions (images) required support from centres which otherwise are dependent on it.

Although Grashey's study has not proved as important for the elucidation of amnesic aphasia without localized lesions as it seemed to be at first, it can still claim lasting value by virtue of several subsidiary findings. It was the first study to investigate the mutual relationship of the speech centres and their dependence on the centre of the sound impressions, and the first to give us a glimpse of the complicated and often devious course of the associations underlying the processes of speech; finally, by proving that reading is accomplished by spelling, it has established the correct approach for the evaluation of the reading disorders. With regard to the last statement, a qualification might be called for. It is probable that for certain types of reading, especially for certain words, the object image of the whole word contributes to its recognition. This explains why persons who are "letter blind",

i.e., unable to read single letters, can nevertheless read their own names or some words very familiar to them, such as the name of a town or a hospital, and why one of Leube's[1] patients was occasionally able to pronounce a word which she had in vain endeavoured to spell, as soon as it was withdrawn from her sight, i.e., as soon as she was stopped in her attempts at spelling it. Leube suggested that in this case the object image of the printed or written word had by then imprinted itself deeply enough to be read as a whole.

Our starting point was a concept of speech disorder according to which some forms of aphasia could be explained solely as the effects of circumscribed lesions of tracts and centres, while the rest could be fully understood as functional changes in the apparatus of speech. We pointed out that in the case of the transcortical motor aphasia the former explanation alone was not applicable and that functional changes had to be assumed for this particular disorder. On the other hand, a critical appraisal of Grashey's case led us to the conclusion that amnesic aphasia could not be explained without the assumption of a localized lesion. We proposed to reconcile these apparently incompatible assumptions by means of the hypothesis that the centres of the speech apparatus reacted as wholes with an alteration of function to partial lesions, and we adopted as such modifications of functions Bastian's three levels of reduction of excitability; i.e., a centre may be (1) no longer excitable at all; (2) excitable only by sensory stimulation; and (3) excitable in association with another centre. We expect to find an interruption of fibre tracts as well as a

[1] Leube: *Ueber eine eigentümliche Form von Alexie* (On a peculiar form of alexia). Zeitsch. f. Klin. Medicin, XVIV, 1889.

modification of function in any given case of speech disorder. The question arises, therefore, which features of an aphasia are attributable to the one and which to the other cause. Our approach calls for a concept of aphasia which is not open to the objections discussed earlier.

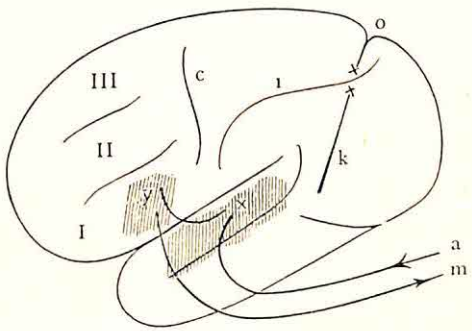
V

In one of the preceding chapters it was pointed out that Wernicke's theory of the speech process was based on a specific assumption concerning the rôle of "centres" in the cerebral cortex, and that clinical observation had failed to bear out certain anticipations which such an assumption would warrant. It appears necessary, therefore, to subject Wernicke's theory to a closer scrutiny. According to him, there are certain fairly well defined areas in the cortex, such as Broca's and Wernicke's areas, the nerve cells of which contain the images (impressions) essential for the process of speech. These images are residues of impressions which reach the brain via the visual or auditory nerves, or which originate as sensations of innervation or of perceptions of movements carried out in the act of speaking. Depending on their origin from one of these sources, they lie together in the cerebral cortex: one area is assumed to contain all "word sound images", another all "glosso-kinaesthetic word images or impressions", etc. These cortical centres are

interconnected by white fibre tracts (association tracts) and in between the centres there is unoccupied cortical territory, i.e., the "functional gaps" according to Meynert. With the latter term we have introduced a part of Meynert's teaching into Wernicke's concepts. Wernicke, who never fails to mention that his theory of aphasia is only an application of Meynert's basic teachings, tended at first to deviate from them in his writings on the speech centres. In his essay on the aphasic syndrome he still regarded the whole of the convolutions around the Sylvian fissure as speech area; later on, however, in his textbook on the diseases of the brain, only parts of the first frontal and first temporal convolutions were presented as speech centres (Fig. 7).

It appears appropriate at this point to consider Meynert's doctrine of the organization and the functions of the brain. My presentation of them here, and the objections raised against them, will be only cursory and sketchy; it cannot possibly do justice to their great importance. However, a full discussion of these theories

FIG. 7



From *Wernicke:* Textbook of the Diseases of the Brain, Vol. 1, p. 206. Fig. XX, schema of the cortical mechanism of speech. *c* central sulcus; *i* interparietal fissure; *o* parieto-occipital fissure; *k* anterior occipital fissure; *I-III* first to third frontal convolutions; xx transitional gyri; *x* sensory speech centre; *y* motor speech centre; *xy* association tract between the two speech centres; *ax* auditory tract; *ym* tract to the speech muscles.

would be beyond the scope of this study which is intended to deal with the concepts of aphasia only. But as the latter cannot be viewed independently of a broad theory of cerebral activity I cannot avoid at least touching upon the general problem of brain function. Meynert's theory of the organization of the brain deserves to be named "cortico-centric". In his far-reaching speculations on anatomical conditions, which are so typical of him, Meynert expressed the view that the cerebral cortex, by its superficial situation, was particularly suited for the reception and retention of all sensory stimuli.[1] He also compared the cerebral cortex to a complex protoplasmic organism which expanded over an object it wanted to incorporate by taking the shape of a cavity.[2] The whole remaining cerebrum thus appeared as an appendix and auxiliary organ of the cerebral cortex, and the whole body as an armour of feelers and tentacles which enabled it to incorporate and to modify the picture of the external world.

According to Meynert, all the fibre tracts of the brain either enter into, or originate from, the cerebral cortex. All the grey masses are interspersed within the course of these tracts on their way to the cerebrum. The spinal cord derives from the cerebral cortex by way of a double origin which a cross section in the region of the cerebral peduncles reveals.[3] The so-called pes pedunculi contains the fibre tracts which convey motor impulses from the cerebral cortex to the periphery as well as the tracts which serve the reception of sensory stimuli in the cortex. In the pes pedunculi, therefore, there is a projection of

[1] Meynert: *Der Bau der Grosshirnrinde* (The organization of the cerebral cortex). Vierteljahrschrift für Psychiatrie, I, 1867.

[2] Idem: *Ibidem*, and "*Psychiatrie*", p. 127.

[3] In speaking of the cortical origin of the spinal cord Meynert referred to efferent as well as afferent tracts. Freud has occasionally followed this usage in this book. (Transl.)

the body insofar as the latter is functionally dependent on the cortex.[1] The so-called tectum pedunculi, on the other hand, conveys to the cortex the knowledge of reflex activities in the spinal cord and brain stem, and with them the first stimuli to spontaneous motor impulses. The grey masses of the brain stem, being linked with the spinal cord as well as with the peripheral sensory apparatus, are either parts of the reflex apparatus which is connected with the cortex through the tectum, or they interrupt the direct cerebral connections as ganglia of the pes pedunculi. The lenticular body belongs to the latter, the corpora quadrigemina and the optic thalami to the former group. The motor tracts through which the skeletal musculature is controlled by the cortex, falls into three divisions which are separated by two nuclei (lentiform-caudate nuclei and grey matter of the anterior horns). In addition, the nuclei pontis and the fibrae arcuatae provide a connection with the cerebellum which is otherwise somewhat neglected in Meynert's map of the brain.

In what way, then, is the body represented in the cerebral cortex which is connected with the periphery by means of these tracts? Meynert calls this representation a "projection", and some of his comments indicate that he actually envisages a projection, i.e., a point by point representation, of the body in the cerebral cortex. This is suggested by the frequent analogy he draws between cerebral cortex and retina. The latter is an end organ which several writers have called an outlying piece of cortex, while morphologically one would expect it to correspond to a piece of spinal grey matter. The following is one of several passages which suggest

[1] Meynert: *Studien über die Bedeutung des zweifachen Rückenmarksursprunges aus dem Gehirn* (Studies on the significance of the double origin of the spinal cord in the cerebrum. Wiener akad. Sitzungsber., LX, Vol. II, 1869.)

that Meynert did think of projection in the strict sense of the term: "It is most improbable that every single fibre tract representing different groups of muscles, areas of the skin, glands and bowels should disperse sufficiently to be represented, by projection, over the whole surface of the cortex";[1] or "a cross section through the cerebral peduncles contains as it were the whole organism though without the senses of smell and sight".[2] However, other parts of Meynert's teachings contradict such an assumption and I hesitate to attribute it to him. However, there can hardly be any doubt that Munk and other workers who have accepted the principles of Meynert's teachings, have more or less explicitly propagated the concept of a complete and topographically exact representation of the body in the cerebral cortex.

I wish to draw attention to the fact that the recent advances in the anatomy of the brain have necessitated considerable changes in Meynert's concept of cerebral organization and have thrown doubt on the role attributed by him to the cortex. In particular, his views on the course of the most important and best known pathway from the cerebral cortex to the skeletal muscles had to undergo far-reaching revisions. The first assumption to be dropped was that of the corpus striatum as a ganglion within the motor tract. The clinicians, under Charcot's leadership, have shown that a lesion of this nucleus affects motor function only because of its vicinity to the internal capsule, while lesions of the ganglion which spare the inner capsule are incapable of causing paralysis. Wernicke[3] has

[1] Meynert: *Bau der Grosshirnrinde* (Organization of the cerebral cortex). l. c., p. 83.

[2] Idem: *Rückenmarksursprung* (Origin of the spinal cord), l. c., p. 488.

[3] Wernicke: *Lehrbuch der Gehirnkrankheiten* (Textbook of the diseases of the brain). 1880 to 1883, Vol. I.

demonstrated that this so-called ganglion of the peduncles lacks any substantial connections with the cerebral cortex. Thus the first internodium had been torn out of the course of Meynert's projection tract. The study of the successive phases of myelinization confirmed Wernicke's view and revealed a new gap in Meynert's concept of the structure of the brain. Flechsig was able to demonstrate that the motor pathway runs without interruption from the cortex through the internal capsule into the peduncles and that there is no connection in the pons between the motor pathway and the cerebellum. The pyramidal tract is now regarded as the direct connection between the anterior horns of the spinal cord and the cortex. Meynert's idea of the relationship of the cerebellum to the motor tract has been dropped. Of the large subcortical nuclei, only the optic thalamus is connected with the cerebral cortex; it is atrophic in cases of congenital malformation of cerebral lobes; the striate body, however, remains intact in cases of lobar degeneration, while it is found to be atrophic in patients with congenital cerebellar atrophy. Thus a formidable portion of the brain, i.e., the corpora striata, the pons and the cerebellum, can be differentiated, as an organ of unknown function, from the rest of the brain with which it has a great many connections, though developmentally and functionally it is fairly independent of it. Although Meynert's hypothesis of the two levels of the cerebral peduncles can no longer be maintained, no alternative hypothesis has so far taken its place. If there be any question of a double origin of the spinal cord, it can only mean one origin in the cortex and thalamus (cortico-thalamic origin) and another which is striato-cerebellar. The whole organization of the brain seems to fall into two central apparatuses of which the cerebral cortex is the younger, while the older one is represented by the

ganglia of the forebrain which have still maintained some of their phylogenetically old original functions. Another important part of Meynert's theory, i.e., the assumption of a double sensory tract, one direct and the other reflectory, seems to have remained unconfirmed. Investigations carried out so far have shown that no fibre tract reaches other parts of the brain without having entered into some connection with the grey matter of the spinal cord or similar structures, and that the reflex tracts always originate from sensory fibres.

Thus the theory of the dominant role of the cerebral cortex has been disproved. On the other hand, some processes previously regarded as sub-cortical, have now been allocated to the cortex. The question arises in what way the body is represented in the cerebral cortex. I believe that the assumption of a projection of the body into the cortex in the strict sense, i.e., of an image that is complete and topographically similar, can be rejected.

I propose to take Henle's concept of the reduction of fibres through grey masses as my point of departure. If one compares the total number of fibres entering the spinal cord from the periphery with that of the fibre tracts leaving the cord to connect it with the brain, the latter proves to be only a fraction of the former. According to Stilling's count, in one instance 807,738 fibres of a nerve root corresponded to no more than 365,814 fibres in a cross section of the upper cervical cord. It follows that the relationship of the spinal cord to the body is different from its relationship to the grey masses of the brain. Only in the spinal cord, and in analogous grey areas, do the prerequisites for a complete projection of the body periphery exist. For each peripheral unit of innervation there exists a corresponding area of grey matter in the spinal cord, and in the extreme case one

single nervous element. Owing to the reduction of the projection fibres through the grey matter of the spinal cord, a unit of grey matter belonging to a higher level can no longer correspond to one peripheral unit, but must be related to several such units. This also holds for the cerebral cortex, and it is therefore appropriate to use different terms for these two types of representation in the central nervous system. If the way in which the periphery is reflected in the spinal cord is called a "projection", its counterpart in the cerebral cortex might suitably be called a "representation", which implies that the periphery of the body is contained in the cerebral cortex not point by point, but through selected fibres, in a less detailed differentiation.

This simple argument requires further elaboration in a different direction; not all the fibres of the highest cross section of the spinal cord serve the connection with the cerebral cortex. A considerable portion, especially those forming the short pathways, exhausts itself within the ventricular grey matter between the nuclei of the medulla, while another portion enters the cerebellum. Only of the pyramidal tract can it be said with certainty that its size in the brain is the same as in the cervical cord, and this tract is no doubt a greatly reduced continuation of the fibres which connect the muscles of the body with the spinal cord. On the other hand, the reduction of the projection fibres is not as great as it might appear from these considerations; if we take the example of the posterior tracts, a certain proportion of their fibres do not reach the cortex, but in their place the latter receives the fibres of the fillet which after various interruptions in the nuclei of the posterior tracts, the oblongata and the thalami, finally represent the posterior tracts in the cerebral cortex. It is not known whether the fibres of the fillet are equal in number to

those of the posterior tracts; probably they are much less numerous. In addition, the cerebrum receives fibres from the cerebellum which could be regarded as an equivalent for the cerebellar connections of the spinal cord. It is therefore quite possible that the cerebral cortex receives at least as many fibres from the periphery, though by devious routes, as were required for projection in the spinal cord.

There is yet another aspect which has not been made sufficiently clear in Meynert's presentation. For Meynert, who in describing pathways is mainly concerned with their cortical connections, a fibre or a fibre tract retains its identity even after having passed through an unlimited number of nuclei. This is indicated by his phrase: "the fibre passes through a grey substance". This naturally gives rise to the impression that the fibre remains the same on its long way to the cortex, apart from the fact that it has possibly entered into several connections. This view can no longer be maintained. If we observe how in the course of individual development myelinization proceeds piecemeal from one nucleus to another, and how for one afferent fibre tract three or more efferent tracts emerge from one single nucleus, these grey masses, and not the fibre tracts, appear to be the organs of the brain. If we follow the course of a sensory afferent tract as far as we know it, and if we regard its frequent interruptions in grey nuclei and its arborizations through them as characteristic,[1] we cannot but assume that the functional significance of a fibre on its way to the cerebral cortex has changed each time it has emerged from a nucleus. Let us take one of the better understood examples; a fibre of the optic nerve conveys a retinal impression to the anterior quadrigeminal body;

[1] See Edinger's, Bechterew's and the present author's investigations into the course of the posterior tracts and of the auditory nerve.

here it terminates,[1] and in its place another fibre goes from the ganglion to the occipital cortex. However, in the grey substance of the quadrigeminal body the retinal impressions become associated with a kinaesthetic oculomotor impression; it is extremely likely that the new fibre between quadrigeminal body and occipital cortex no longer conveys a retinal stimulus, but the association of one or more such impressions with kinaesthetic impressions. The complexity of this change in the functional significance of the fibres must be even greater in the fibre systems serving skin and muscle sensations; we are still quite ignorant of the elements which contribute to the functional changes that conducted stimuli undergo. We can only presume that the fibre tracts, which reach the cerebral cortex after their passage through other grey masses, have maintained some relationship to the periphery of the body, but no longer reflect a topographically exact image of it. They contain the body periphery in the same way as—to borrow an example from the subject with which we are concerned here—a poem contains the alphabet, i.e., in a completely different arrangement serving other purposes, in manifold associations of the individual elements, whereby some may be represented several times, others not at all. If it were possible to follow in detail the rearrangement which takes place between the spinal projection and the cerebral cortex, one would probably find that the underlying principle is purely functional, and that the topographic relations are maintained only as long as they fit in with the claims of function. As there is no indication that this rearrangement

[1] See Darkschewitsch: *Ueber die sogenannten primären Opticuscentren und ihre Beziehung zur Grosshirnrinde* (Investigations on the so-called primary centres of the optic nerve and their relationship to the cerebral cortex). Arch. f. Anat. u. Phys., 1886.

is reversed in the cerebral cortex to produce a topographically complete projection, we may suppose that the representation of the body periphery in the higher parts of the brain, and also in the cortex, is no longer topographical but only functional. The animal experiment is bound to obscure this fact because it cannot reveal any other but a topographical relationship. However, I believe that those who seriously look for a cortical centre for the musculus extensor pollicis longus, or for the musculus rectus oculi, or for the sensibility of a certain area of the skin, are labouring under a misconception of the function of the cortex, as well as of the complicated conditions which make this function possible.[1]

After this digression we now return to the problem of aphasia. We remember that, under the influence of Meynert's teachings, the theory has been evolved that the speech apparatus consists of distinct cortical centres; their cells are supposed to contain the word images (word concepts or word impressions); these centres are said to be separated by functionless cortical territory, and linked to each other by the association tracts. One may first of all raise the question as to whether such an assumption is at all correct, and even permissible. I do not believe it to be.

Considering the tendency of earlier medical periods to localize whole mental faculties, such as are defined in psychological terminology, in certain areas of the brain, it was bound to appear as a great advance when Wernicke declared that only the simplest psychic elements, i.e., the various sensory perceptions, could be

[1] I should like to point out that this concept of the cortical representation of the body challenges Munk's views of the point to point projection of the retina in the occipital lobe, and that a study of the cortical hemianopias ought to confirm or refute it.

localized in the cortex, the areas concerned being those of the central terminations of the sensory nerves. But does one not in principle make the same mistake, irrespective of whether one tries to localize a complicated concept, a whole mental faculty or a psychic element? Is it justified to immerse a nerve fibre, which over the whole length of its course has been only a physiological structure subject to physiological modifications, with its end in the psyche and to furnish this end with an idea or a memory? Now that "will" and "intelligence", etc., have been recognized as psychological technical terms referring to very complicated physiological states, can one be quite sure that the "simple sensory impression" be anything else but another such technical term?

The relationship between the chain of physiological events in the nervous system and the mental processes is probably not one of cause and effect. The former do not cease when the latter set in; they tend to continue, but, from a certain moment, a mental phenomenon corresponds to each part of the chain, or to several parts. The psychic is, therefore, a process parallel to the physiological, "a dependent concomitant".

I am well aware that the writers whose views I am opposing here cannot have been guilty of thoughtless mistakes in their scientific approach. They obviously mean only that the physiological modification of the nerve fibre through sensory stimuli produces another modification in the central nerve cells which then becomes the physiological correlate of the "concept" or "idea". As they know a lot more about ideas than of the physiological modifications, which are still undefined and unknown, they use the elliptic phrase: an idea is localized in the nerve cell. Yet this substitution at once leads to a confusion of the two processes which need have nothing in common with each other. In

psychology the simple idea is to us something elementary which we can clearly differentiate from its connection with other ideas. This is why we are tempted to assume that its physiological correlate, i.e., the modification of the nerve cells which originates from the stimulation of the nerve fibres, be also something simple and localizable. Such an inference is, of course, entirely unwarranted; the qualities of this modification have to be established for themselves and independently of their psychological concomitants.[1]

What then is the physiological correlate of the simple idea emerging or re-emerging? Obviously nothing static, but something in the nature of a process. This process is not incompatible with localization. It starts at a specific point in the cortex and from there spreads over the whole cortex and along certain pathways. When this event has taken place it leaves behind a modification, with the possibility of a memory, in the part of the cortex affected. It is very doubtful whether this physiological event is in any way associated with something psychic. Our consciousness contains nothing that would, from the psychological point of view, justify the term "latent memory image". Yet whenever the same cortical state is elicited again, the previous psychic event re-emerges as a memory. We have, of course, not the slightest idea how animal tissue can possibly undergo, and differentiate, so many various modifications. But that it is able to do so is proved by the example of the spermatozoa in which the most varied and highly

[1] Hughlings Jackson has most emphatically warned against such a confusion of the physical with the psychic in the study of speech: "In all our studies of diseases of the nervous system we must be on our guard against the fallacy that what are physical states in lower centres fine away into psychical states in higher centres; that for example, vibrations of sensory nerves become sensations, or that somehow or another an idea produces a movement." Brain I, p. 306.

differentiated modifications lie dormant and ready to develop.

Is it possible, then, to differentiate the part of "perception" from that of "association" in the concomitant physiological process? Obviously not. "Perception" and "association" are terms by which we describe different aspects of the same process. But we know that the phenomena to which these terms refer are abstractions from a unitary and indivisible process. We cannot have a perception without immediately associating it; however sharply we may separate the two concepts, in reality they belong to one single process which, starting from one point, spreads over the whole cortex. The localization of the physiological correlates for perception and association is, therefore, identical, and as localization of a perception means nothing else but localization of its correlate, we cannot possibly have a separate cortical localization for each. Both arise from the same place and are nowhere static.

With this refutation of a separate localization for ideation and association of ideas we have disposed of an important reason for differentiating between centres and pathways of speech. In every part of the cortex serving speech we have to assume similar functional processes, and we have no need to call on white fibre tracts for the association of ideas within the cortex.

There is a post-mortem finding which proves that the association of ideas takes place through the fibres situated in the cortex itself. I am again referring to Heubner's case which has already taught us an important lesson. Heubner's patient showed the form of speech disorder called by Lichtheim transcortical sensory aphasia and attributed by him to interruption of the fibre tracts from the sensory speech centre to the areas serving the association of concepts. According to his theory a lesion in the

white matter underlying the sensory centre was to be expected in this case. Instead, post-mortem examination revealed a superficial cortical softening which separated the otherwise intact sensory centre, which was functioning normally, from most of its cortical connections outside the speech area. Heubner did not fail to emphasize the importance of this finding, and Pick[1] drew the same conclusions from it as we do, i.e., that the speech association tracts seem to go through the cortex itself. This does not, of course, exclude the possibility of subcortical association tracts contributing to this function.

Our conception of the speech apparatus will undergo a thorough transformation when we finally consider the third stipulation of the Meynert-Wernicke theory, i.e., that the areas functioning as speech centres are separated by "functionless gaps". A statement such as this, apparently based on morbid anatomy, seems at first unassailable. However, if one examines the way in which distinct centres were inferred from post-mortem findings, it becomes clear that morbid anatomy is incapable of deciding this question. One only needs to glance at the diagram in which Naunyn entered the extent of the lesions in seventy-one cases of aphasia. The areas which showed the greatest overlapping of lesions were regarded as the speech centres, i.e., as the areas the intactness of which was indispensable for speech to function normally. There might, however, be other cortical areas also serving speech, though their destruction can be tolerated more easily. If there are such areas we shall not be able to detect them from the study of Naunyn's tables. Possibly a speech disorder caused by lesion of areas other

[1] Pick: *Ueber die sogenannte Re-evolution (Hughlings Jackson) nach epileptischen Anfällen nebst Bemerkungen über transitorische Worttaubheit* (On the so-called re-evolution [Hughlings Jackson] following epileptic seizures, with observations on transitory word deafness). Arch. f. Psychiat., XXII, 1891.

than the speech centres, may be due to a remote effect which such lesions exert on the centres; it is also possible that the areas which in the table appear less frequently involved, are also speech centres but not indispensable and constant ones.

Let us now turn to the question which functions have been allocated to the so-called functionless cortex between and beside the speech centres? Meynert states quite clearly (*Psychiatrie*, p. 140): "It follows that in the physiological process of the occupation of the cerebral cortex by memory images an increasing number of cortical cells are thus involved; on this process the growth of the child's range of imagery depends. Very probably the receptivity of memory, which is the basis of all intellectual achievements, is limited by the number of cortical cells available." The latter sentence can be taken to mean that not only the intellectual development in childhood but also the acquisition of later knowledge, such as the learning of a new language, depends on the occupation of hitherto unoccupied cortical areas, similar to the way in which a town expands by people settling in areas outside its walls.

In an earlier note, Meynert had allocated the task of taking over the functions of the speech centres, in the case of experimental or other lesions, to the unoccupied areas within their neighbourhood. This theory derived support from experiments carried out by Munk whose hypotheses had been based entirely on Meynert's teachings.

Having revealed the trend underlying the assumption of the "functionless gaps" in the cerebral cortex we can now proceed to examine its usefulness for the understanding of the aphasias. In doing so we find that the exact opposite of what would have to be expected according to the above theory, actually takes place. The

function of speech presents the most perfect examples of new acquisitions. Learning to read and to write is one of them, compared with the primary activity of speaking; this acquisition can be impaired by lesions localized outside the speech centres because additional sensory functions, i.e., the visual and the cheiro-motor, are involved in these activities. But if I learn to understand and to speak several foreign languages, or acquire the knowledge of the Greek or Hebrew alphabet in addition to the one I learned first, or practise shorthand and other forms of writing beside my cursive handwriting, I am acquiring abilities which may demand memories many times more numerous than the original language; all these new acquisitions of the faculty of speech are obviously located in the same areas which we know as the centres for the first learned language. It never happens that an organic lesion causes an impairment affecting the mother tongue and not a later acquired language. If in the case of a German who understands French, the word sounds of the latter language had a different localization from the German word sounds, it ought to happen occasionally that following a cerebral softening such a patient would cease to understand German while still understanding French. In fact, the opposite is invariably the case, and this applies to all functions of speech. In reviewing relevant case material which, unfortunately, is much too small considering its great theoretical interest, I could find only two factors determining the character of the speech disorders in the polyglot; (1) the influence of the age of the acquisition, and (2) that of practice. These two factors work as a rule in the same direction. It is noteworthy that if they differ, the earlier acquired language may survive the one in greater use. But their relationship can never be explained by an unusual localization but only by the

two functional factors mentioned. Obviously a new set of associations is capable of being superimposed on the established associations involved in speaking; we are clearly aware of this process as long as the new associations can be carried out only with difficulty. The superimposed set of associations is impaired before the primary one, wherever the site of the lesion. Perhaps there is no better illustration of the degree to which a moderately severe modification of the speech apparatus can persist in spite of damage, contrary to all theories of localization of ideas, than the following which I am borrowing from Hughlings Jackson. This author, on whose views I have based almost all the arguments which I have advanced in refuting the localizatory theory of the aphasias, discussed the not unusual case of the motor aphasic who, apart from "yes" or "no", has retained a residue of speech which otherwise would represent a complex activity of language. This residue frequently consists of a vigorous curse (*sacré nom de dieu*, Goddam, etc.); Hughlings Jackson points out that even in normal persons such an utterance belongs to the emotional and not to the intellectual language. In other cases, however, this recurrent utterance is not a curse, but a phrase of special significance; one might regard it as very amazing indeed that exactly these cells or these memory traces should have escaped the general destruction. Some of these cases, however, permitted a very plausible interpretation. For instance, a man who could say only "I want protection", owed his aphasia to a fight in which he had been knocked unconscious by a blow on the head. Another patient had the curious speech remnant: "List complete": he was a clerk who had a stroke immediately after he had laboriously completed a catalogue. Such instances suggest that these utterances are the last words produced by the speech apparatus before injury, or even

at a time when there already existed an awareness of the impending disability. I am inclined to explain the persistence of these last modifications by their intensity if they happen at a moment of great inner excitement. I remember having twice been in danger of my life, and each time the awareness of the danger occurred to me quite suddenly. On both occasions I felt "This is the end", and while otherwise my inner language proceeds with only indistinct sound images and slight lip movements, in these situations of danger I heard these words as if somebody was shouting them into my ears, and at the same time I saw them as if they were printed on a piece of paper floating in the air.

We have rejected the assumptions that the speech apparatus consists of distinct centres separated by functionless areas, and that ideas (memories) serving speech are stored in certain parts of the cortex called centres while their association is provided exclusively by subcortical fibre tracts. It only remains for us to state the view that the speech area is a continuous cortical region within which the associations and transmissions underlying the speech functions are taking place; they are of a complexity beyond comprehension.

How can such a theory explain the existence of the speech centres, especially of the areas of Broca and Wernicke established by morbid anatomy? A glance at the convexity of a left hemisphere will enlighten us; the situation of the so-called speech centres suggests an explanation which fits well into our theory. These centres lie far apart; according to Naunyn, they are situated in the posterior part of the first temporal convolution, the posterior part of the third frontal gyrus and in the inferior part of the parietal lobe where the angular gyrus merges into the occipital lobe; the

site of a fourth centre, for writing, does not seem to be definitely established (?posterior part of the middle frontal gyrus). These areas are situated in such a way that there lies between them a large cortical region, i.e., the insula with the convolutions covering it; the lesion of any part of this area is probably always associated with speech disorder. Although the extent of this area cannot be exactly delineated from a survey of lesions found in cases of aphasia, one can nevertheless say that the so-called speech centres form the outlying parts of the speech area assumed by us, and that speech disorders occur when lesions are situated within the external boundaries of these centres, i.e., towards the centre of the hemisphere, while lesions in cortical areas lying outside them are of a different significance. Thus the "centres" appear as the corner stones of the speech territory. Next we have to consider the areas adjoining these centres from outside. Broca's area is immediately adjacent to the centres of the bulbar motor nerves. Wernicke's area is situated in a region which also contains the acoustic termination, the exact localization of which is unknown; the visual speech centre borders on those parts of the occipital lobe in which we know the optic nerve to terminate. An arrangement such as this, though meaningless from the point of view of the centre theory, has for us the following significance:

The association area of speech, into which visual, auditory, and motor (or kinaesthetic) elements enter, extends for that very reason between the cortical areas of those sensory nerves and the motor regions concerned with speech. If we now imagine a movable lesion of constant size within this association area, its effect will be the greater the more it approaches one of these cortical fields, i.e., the more peripherally it is situated within the speech area. If it borders immediately on one of these

cortical fields it will cut off the association area from one of its tributaries, i.e., the mechanisms of speech will be deprived of the visual, or auditory, or some other element, as every association of that nature used to come from that particular cortical field. If the lesion is moved towards the interior of the association area its effect will be more indefinite; in no event will it be able to destroy all possibilities of one particular category of associations. Thus the parts of the speech region bordering on the cortical fields of the optic, auditory and motor cranial nerves have gained the significance demonstrated by morbid anatomy which has established them as centres of speech. This significance, however, holds only for the pathology, and not for the physiology of the speech apparatus, because it cannot be maintained that in these parts other, or more important, processes take place than in those parts of the speech area the damage of which is better tolerated. This view follows directly from our refusal to separate the process of the idea (concept) from that of association, and to localize the two in separate parts.

Wernicke came near to these views only in his last observations on this subject when he expressed doubt as to whether we were justified in assuming separate centres for reading within the visual receptive area of the cortex, and for writing within the so-called motor arm-region (l. c. p. 477). But his doubt was not of a fundamental nature; it amounted to no more than an anatomical amendment implying that the visual and cheiro-motor impressions so important for speech, were situated among other impressions contributing to speech. Heubner, on the contrary, in discussing his case felt compelled to ask a question similar to the one raised here for speech in general: "Are there perhaps no cortical areas for mind blindness, mind deafness and mind muteness? Are

these symptoms not rather simply due to the separation of the cortical areas subserving these functions from the rest of the cortex by lesions localized in their vicinity?"

There are two possible objections against the validity of our views about the centres:

(1) If destruction of the parts of the speech area bordering directly on a cortical receptive or motor field has the described effect on the speech function only because it has severed the connections with the respective sources of association, the destruction of these receptive and cortical areas themselves ought to have the same result. This, however, is contrary to clinical experience which has established that all such lesions cause localized symptoms without speech disorder. This first objection can easily be disposed of if one considers that all these cortical areas are bilateral, while that of the association area of speech is organized in one hemisphere only. Destruction of one visual cortical area, for example, will not interfere with the utilization of visual stimuli for speech, i.e., with reading, because the speech area retains its connections with the contralateral visual cortex which, in this particular case, is provided by crossed white fibres. If, however, the lesion moves to the boundary of the visual receptive area, alexia ensues, probably because the connection not only with the homolateral, but also with the contralateral visual area has been severed. We therefore have to add to our theory: the appearance of centres is also created by the fact that the fibres from the cortical receptive fields of the other hemisphere enter at the same place, i.e., on the periphery of the speech area where, in case of lesion, the connection with the homolateral receptive areas is also effected. This is plausible, because for the function of speech association the presence of a bilateral origin

of visual, auditory and other stimuli is physiologically irrelevant.

The assumption, by the way, that the speech region is connected with cortical areas of both hemispheres is not new but has been taken over from the theory of the centres. The precise anatomy of these crossed connections has not yet been established, but when it is known it might explain some peculiarities in the localization and extent of the so-called centres, as well as some of the individual features of the speech disorders.

(2) The question may be raised what advantage there be in denying the existence of special centres for speech while we have to assume cortical fields, i.e., centres, for the visual and auditory nerves, and for the motor organs of speech. The answer is that there is no reason why these areas should not be subjected to similar considerations. However, their existence cannot be disputed; their extent is defined by the anatomical fact of the termination of the sensory nerves and the origin of the pyramidal tract in circumscribed areas of the cortex. The region of speech associations, however, lacks these direct relations to the periphery of the body. It certainly has no sensory and most probably no special motor "projection fibres".[1]

[1] I reported the main contents of this study in a paper read to the "Wiener physiologischer Club" (Vienna Physiological Club) as early as 1886. However, the statutes of this club do not allow for a claim to priority to be based on its proceedings. In 1887 Nothnagel and Naunyn presented their well-known review on the localization of brain diseases to the Congress of Internal Medicine at Wiesbaden. Their views agree with those presented here in several important points. Nothnagel's observations on the concept of cerebral centres as well as Naunyn's remarks on the topography of the speech areas are likely to make readers suspect that my study was influenced by their highly significant review. This was not the case; the stimulus to this study came, in fact, from papers published by Exner jointly with my late friend Josef Paneth in Pflüger's Archiv.

VI

Our concept of the organization of the central apparatus of speech is that of a continuous cortical region occupying the space between the terminations of the optic and acoustic nerves and of the areas of the cranial and certain peripheral motor nerves in the left hemisphere. It probably covers, therefore, the same area which Wernicke was inclined to allocate to speech in his first paper, i.e., all the convolutions forming the Sylvian fissure. We have refused to localize the psychic elements of the speech process in specified areas within this region; we have rejected the supposition that there were areas within this region which were excluded from the speech function in general and kept in reserve for the acquisition of new knowledge of speech. Finally, we have attributed the fact that pathology has demonstrated centres of speech, though of indefinite delimitation, to the situation of the adjoining receptive and motor cortical areas and of the crossed fibre tracts. Thus the speech centres are, in our view, parts of the cortex which may claim a pathological but no special physiological significance. We feel justified in rejecting the differentiation between the so-called centre or cortical aphasias and the conduction (association) aphasias, and we maintain that all aphasias originate in interruption of associations, i.e., of conduction. Aphasia through

destruction or lesion of a centre is to us no more and no less than aphasia through lesion of those association fibres which meet in that nodal point called a centre.

We have also asserted that every aphasia is directly, or through some remote effect, caused by disturbance within the cortex itself. This implies that the speech area has no afferent or efferent pathways of its own extending to the periphery of the body. This statement is proved by the fact that subcortical lesions of any location are incapable of producing aphasia, provided anarthria is excluded by definition. Nobody has ever been known to become word deaf as the result of a lesion in the auditory nerve, in the medulla oblongata, in the posterior corpora quadrigemina or in the internal capsule unless he had been deaf already; nor has anybody ever been made word blind by a partial lesion of the optic nerve, or of the diencephalon, etc. However, Lichtheim differentiates a subcortical word deafness and a subcortical motor aphasia, and Wernicke postulates subcortical alexia and agraphia. They do not attribute these types of speech disorder to lesions of subcortical fascicles of association fibres, which in our view cannot be differentiated from association fibres within the cortex itself, but to lesions of radial, i.e., afferent or efferent speech tracts. It is therefore necessary to analyse these subcortical aphasias more precisely.

The characteristic features of a subcortical sensory aphasia can easily be deduced from Lichtheim's schema which postulates a special auditory tract αA (Fig. 3) for speech. The patient is supposed to be unable to perceive word sounds, yet capable of availing himself of previously acquired sound impressions and of carrying out all other speech functions faultlessly. Lichtheim actually found such a case; although the early stages of

this patient's illness had not been fully elucidated, his final state entirely conformed to the picture supposed to be caused by interruption of αA. I confess that in view of the importance of the sound images for the speech function I have found it exceedingly difficult to find another explanation for this subcortical sensory aphasia which would make the assumption of an afferent auditory tract αA unnecessary. I was already inclined to explain Lichtheim's case by assuming that individual speech might be independent of the sound images; the patient was a highly educated journalist. But such an explanation would quite rightly have been regarded as a mere subterfuge.

I therefore searched the literature for similar cases. Wernicke, in reviewing Lichtheim's paper, stated that he had made a similar observation which he was going to publish in the regular reports from his Klinic. Unfortunately I have not been able to find this report in the literature.[1] However, I found a case described by Giraudeau[2] which closely resembled Lichtheim's patient. Giraudeau's patient (Bouquinet) was able to speak perfectly well but she showed a severe word deafness without being deaf. However, the data concerning her hearing ability were incomplete. She could understand questions addressed to her but only after they had been repeated several times, and even then she failed frequently. Once a question had been understood and answered, all following replies would continue in the same train of thought, the patient not taking any notice of later questions. The two patients appear even more alike if we consider that the behaviour of Lichtheim's patient differed from that commonly observed in cases of

[1] A private enquiry at the Breslau Klinik revealed that the cases mentioned in this context by Wernicke have not yet been published.

[2] Giraudeau: *Revue de médecine*, 1882; also quoted by Bernard, l. c.

word deafness. He did not make any effort to understand questions addressed to him; he gave no reply nor did he appear to pay any attention to what he heard. Perhaps the patient, by this apparently purposive behaviour, gave the wrong impression of being completely word deaf, while possibly repeated and urgent requests might have made him, like Bouquinet, understand. Word deaf patients, as a rule, perceive language which they are unable to understand; they believe, however, that they have understood something and, as a result, tend to give inappropriate answers.

The post-mortem examination of Giraudeau's patient revealed a lesion of the first and second temporal convolution, such as has so frequently been found to underlie ordinary sensory aphasia. Nobody, looking at the drawing attached to Giraudeau's communication, would have thought that this lesion had caused anything but the common form of sensory aphasia. But there is another aspect to be considered. The lesion in Giraudeau's case was again an unusual one, i.e., a tumour (gliosarcoma). In discussing transcortical motor aphasia, I ventured the opinion that probably a lesion of the speech apparatus did not only cause localizing signs, but that the special nature of the disease process might be revealed by a functional modification of its symptoms. Giraudeau's case, therefore, does not prove the existence of the subcortical fibre tract αA. The tumour found at the post-mortem examination had not proliferated from the white matter outwardly, having perhaps in an earlier state caused a subcortical lesion only. On the contrary, it was attached to the meninges and could easily be lifted from the softened white matter. I therefore feel justified in assuming that the subcortical sensory aphasia is not due to a lesion of a subcortical pathway αA, but to damage of the same localization

as found in cases with cortical sensory aphasia. However, I am unable to throw light on the specific functional state of the area thus affected.[1]

Subcortical motor aphasia can be dealt with more briefly. According to Lichtheim it is characterized by intact writing ability in the presence of symptoms of

[1] Notwithstanding these considerations, I still find it very difficult to explain the subcortical sensory aphasia, i.e., word deafness without impairment of spontaneous speech, while Lichtheim's schema disposes of the problem by simply assuming interruption of a tract αA. It was therefore of great value to me to come across a paper by Adler in which a similar case was described as "a combination of subcortical and transcortical sensory aphasia" (*Beitrag zur Kenntnis der selteneren Formen der sensorischen Aphasie.* [A contribution to the knowledge of the rarer forms of sensory aphasia.] Neurol. Centralblatt, May 15 and June 1, 1891.)

A comparison of Adler's case with those of Lichtheim and Wernicke contributes to the understanding of the so-called subcortical sensory aphasia. Two points in particular are illuminating. (1) Lichtheim mentioned the possibility that his patient may have been slightly deaf as the data about his hearing ability were incomplete. Wernicke's patient had a defect for higher tones. Adler's patient had definitely diminished hearing, which according to the author was most probably due to a disturbance in the apparatus of sound conduction. It is, therefore, possible that ordinary deafness, peripheral or central in origin, may play a part in this disorder, such as was the case in Arnaud's patients to be referred to later. (2) More decisive still is the following conformity which can hardly be incidental. Both cases (Lichtheim and Adler; Wernicke's brief note is silent on this point) developed the picture of subcortical sensory aphasia only after repeated cerebral accidents of which at least one had involved the minor hemisphere; Lichtheim's patient had a left-sided facial palsy, Adler's case a left-sided hemiplegia. Adler mentioned this coincidence without recognizing its significance for the explanation of pure word deafness. I feel justified in assuming that subcortical sensory aphasia is caused not, as postulated in Lichtheim's schema, by a simple tract interruption, but through incomplete bilateral lesions in the receptive field of hearing, perhaps combined with peripheral deafness, as was the case in Arnaud's patients. Such complicated conditions for an apparently simple speech disorder fit better into my conception of the sensory aphasias than into that of Lichtheim.

cortical motor aphasia. Wernicke, who made a careful analysis of the disorders of written language, refused to accept this criterion. To him the one characteristic feature of the subcortical motor aphasia is the patient's ability to state the number of syllables. The controversies over Lichtheim's test have been mentioned earlier in this book. Some observations made by Dejerine[1] have in the meantime confirmed the significance of Lichtheim's syllable test for the diagnosis of subcortical motor aphasia. However, this particular speech disorder could with equal justification be classified as anarthria rather than aphasia.

Several well observed cases, the most recent one by Eisenlohr,[2] suggest that damage underneath Broca's area causes a speech disorder which can be described as literal paraphasia and which represents a transition to dysarthria. For the motor part of the speech apparatus alone, therefore, a special pathway to the periphery may have to be conceded. However, in attributing a special efferent tract to the motor speech area, we want to point out that the deeper the lesion is situated the more closely the disability resembles anarthria. Aphasia still remains a cortical phenomenon.

Therefore, the speech apparatus as conceived by us, has no afferent or efferent pathways of its own, except for a fibre tract the lesion of which causes dysarthria. We shall refer to the so-called subcortical reading and writing disorders later on.

We now propose to inquire what kind of hypotheses have to be made about the causation of aphasias following

[1] Dejerine: *Contribution à l'étude de l'aphasie motrice sous-corticale et de la localisation cérébrale des centres larynges (muscles phonateurs).* (A contribution to the study of the subcortical motor aphasia and to the cerebral localization of the centres of the speech muscles.) Compt. rend. de la Soc. de Biologie, 1891, No. 8.

[2] Eisenlohr: l. c.

lesions of a speech apparatus thus organized; or, in other words, what does the study of the aphasias teach us about the function of this apparatus? In doing this we shall endeavour to separate the psychological from the anatomical aspect of the problem as much as possible.

From the psychological point of view the "word" is the functional unit of speech; it is a complex concept constituted of auditory, visual and kinaesthetic elements. We owe the knowledge of this structure to pathology which demonstrates that organic lesions affecting the speech apparatus result in a disintegration of speech corresponding to such a constitution. We have learned to regard the loss of any one of these elements as the most important pointer to the localization of the damage. Four constituents of the word concept are usually listed: the "sound image" or "sound impression", the "visual letter image", the "glosso-kinaesthetic and the cheiro-kinaesthetic images or impressions". However, this constitution appears even more complicated if one considers the probable process of association involved in the various speech activities.

(1) We learn to speak by associating a "word sound image" with an "impression of word innervation". When we have spoken we are in possession of a "kinaesthetic word image", i.e., of the sensory impressions from the organs of speech. The motor aspect of the "word" therefore is doubly determined. Of its two elements the former, i.e., the impression of word innervation, seems to be the least important psychologically. Its existence as a psychological element may even be disputed. We also perceive, after having spoken, a "sound image" or "sound impression" of the spoken word. As long as we have not perfected our speech, the second sound image, though associated with the first, need not be identical with it. At this stage, which

is the phase of speech development in childhood, we use a language built up by ourselves; in associating various other word sounds with the one produced by ourselves we behave like the motor aphasics.

(2) We learn the language of others by endeavouring to equate the sound image produced by ourselves as much as possible to the one which had served as the stimulus for the act of innervation of our speech muscles, i.e., we learn to "repeat". In "continuous speech" we produce a series of words by waiting with the innervation of the speech muscles until the word sound, or the kinaesthetic word impression of the preceding word, or both, have been perceived. The safeguards of our speech against breakdown thus appear over-determined, and it can easily stand the loss of one or the other element. However, the loss of the correcting function of the second sound image and of the kinaesthetic word image explains some peculiarities of paraphasia, both physiological and pathological.

(3) We learn to spell by associating the visual images of the letters with new sound images which inevitably recall word sounds already known. We immediately repeat the word sound characteristic of the letter. Thus, in spelling aloud, the letter, too, appears determined by two sound impressions which tend to be identical, and two motor impressions which closely correspond to each other.

(4) We learn to read by linking up with each other, according to certain rules, a succession of word innervation impressions and kinaesthetic word impressions perceived in enunciating individual letters. As a result, new kinaesthetic word images originate, but as soon as they have been enunciated we detect from their sound images that both kinaesthetic and sound images so perceived have long been familiar to us, being identical

with those used in speaking. Next, we associate with those word images acquired by spelling the significance attached to the original word sounds. Now we read with understanding. If we have originally spoken a dialect instead of a literary language, we have to super-associate the kinaesthetic and sound impressions perceived in spelling aloud over the original ones, and we have to acquire a new language in this way; this process is facilitated by the resemblance between dialect and literary language.

This presentation shows that the process of learning to read is very complicated indeed and entails a frequent shift of the direction of the associations. It also suggests that the defects of reading in aphasia originate in various ways. Impairment of reading letters is characteristic of a defect of the visual element. The assembling of letters to a word takes place in the process of transmission to the speech tract; it will therefore be abolished in motor aphasia. The understanding of what has been read is effected only with the aid of the sound images produced by the words uttered, or through the kinaesthetic impressions produced in speaking. Reading with understanding thus proves to be a function which disintegrates as the result not only of motor but also of auditory defects, furthermore a function which is independent of the act of reading itself. Everybody knows from self observation that there are several kinds of reading some of which proceed without understanding. When I read proofs with the intention of paying special attention to the letters and other symbols, the meaning of what I am reading escapes me to such a degree that I require a second perusal for the purpose of correcting the style. If, on the other hand, I read a novel, which holds my interest, I overlook all misprints and it may happen that I retain nothing of the names of the persons

figuring in the book except for some meaningless feature, or perhaps the recollection that they were long or short, and that they contained an unusual letter such as x or z. Again, when I have to recite, whereby I have to pay special attention to the sound impressions of my words and to the intervals between them, I am in danger of caring too little about the meaning, and as soon as fatigue sets in I am reading in such a way that the listener can still understand, but I myself no longer know what I have been reading. These are phenomena of divided attention which are of particular importance here, because the understanding of what is read takes place over circuitous routes. It is clear from the analogy with our own behaviour that understanding becomes impossible once reading itself has become difficult, and we must beware of regarding this as an indication of a lesion in a fibre tract. Reading aloud is not to be regarded as a different function from reading to oneself, except that it tends to distract attention from the sensory part of the reading process.

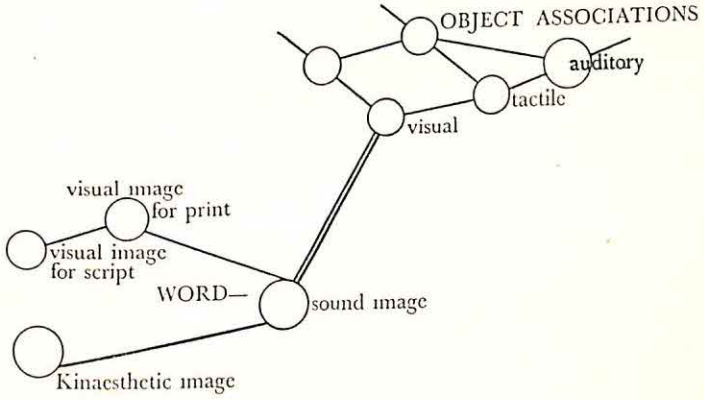
(5) We learn to write by reproducing the visual images of the letters with the help of kinaesthetic impressions received from the hand (cheiro-kinaesthetic impressions) until we have obtained identical or similar pictures. As a rule, the pictures produced in writing are only similar to, and super-associated over those perceived in reading, as we learn to read print but have to use different characters in handwriting. Writing is comparatively simpler and less vulnerable than reading.

(6) It can be assumed that the various speech activities continue to be performed by way of the same associations by which we learned them. Abbreviations and substitutions may be employed, but their nature is not always easy to recognize. Their significance is still further reduced by the consideration that in cases of

organic lesion the speech apparatus as a whole probably suffers some damage and is forced into a return towards the primary and secure, though more cumbersome modes of associations. In the case of the experienced reader the influence of the "visual word image" makes itself felt, with the result that single words, especially proper names, can be read even without recourse to spelling.

The word, then, is a complicated concept built up from various impressions, i.e., it corresponds to an intricate process of associations entered into by elements of visual, acoustic and kinaesthetic origins. However,

FIG. 8



Psychological schema of the word concept.
The word concept appears as a closed complex of images, the object concept as an open one. The word concept is linked to the concept of the object via the sound image only. Among the object associations, the visual ones play a part similar to that played by the sound image among the word associations. The connections of the word sound image with object associations other than the visual are not presented in this schema.

the word acquires its significance through its association with the "idea (concept) of the object", at least if we restrict our considerations to nouns. The idea, or concept, of the object is itself another complex of associations composed of the most varied visual, auditory, tactile,

kinaesthetic and other impressions. According to philosophical teaching, the idea of the object contains nothing else; the appearance of a "thing", the "properties" of which are conveyed to us by our senses, originates only from the fact that in enumerating the sensory impressions perceived from an object, we allow for the possibility of a large series of new impressions being added to the chain of associations (J. S. Mill[1]). This is why the idea of the object does not appear to us as closed, and indeed hardly as closable, while the word concept appears to us as something that is closed though capable of extension.

In the light of observations in speech disorders we have formed the view that the word concept (the idea of the word) is linked with its sensory part, in particular through its sound impressions, to the object concept. In consequence, we have arrived at a division of speech disorders into two classes: (1) verbal aphasia, in which only the associations between the single elements of the word concept are disturbed; and (2) asymbolic aphasia, in which the association between word concept and object concept are disturbed.

I am using the term asymbolia in a different sense from that given to it by Finkelnburg[2] because "asymbolic" seems more appropriate a designation for the relationship between the word and the idea of the object than for that between the object and its idea. For disturbances in the recognition of objects, which Finkelnburg called asymbolia, I should like to propose the term "agnosia". It is quite possible that agnostic disturbances which occur

[1] J. S. Mill: *Logic*, I, Chap. III, and "An examination of Sir William Hamilton's philosophy".

[2] Quoted from Spamer: *Ueber Aphasie und Asymbolie, nebst Versuch einer Theorie der Sprachbildung* (On aphasia and asymbolia; with a tentative theory of the development of speech). Archiv. f. Psychiatrie, VI, 1876.

only in cases of bilateral and extensive cortical lesions, may also entail a disturbance of speech as all stimuli to spontaneous speech arise from object associations. Such speech disorders I should call the third group of aphasias, or "agnostic aphasias". Clinical experience has in fact acquainted us with several cases which call for such a concept.

The first case of agnostic aphasia is that of Farges[1] which was inadequately observed and most inappropriately interpreted as "*aphasie chez une tactile*"; but I hope the clinical facts will speak for themselves. The patient was a case of cerebral blindness, probably due to bilateral cortical lesions. She did not reply when addressed, and when one tried hard to contact her she kept on repeating, "*Je ne veux pas, je ne peux pas!*" in a tone of extreme impatience. She was unable to recognize her doctor by his voice. However, as soon as he felt her pulse, i.e., as soon as he provided her with the opportunity of a tactile association, she at once recognized him, called him by his name and chatted with him without any sign of aphasia, until he let her hand go and thus again became inaccessible to her. The same happened in relationship to objects when she was given the opportunity of producing associations of touch, smell or taste by being offered the respective sensory stimuli. As long as they lasted she had the necessary words at her command and behaved in a purposeful manner; however, as soon as she was deprived of them she resumed her monotonous expressions of impatience or uttered incoherent syllables and proved unable to understand what was said to her. This patient therefore had a completely intact speech apparatus which she was unable to utilize unless it was stimulated by those object associations which had remained intact.

[1] Farges: *Aphasie chez une tactile* (Aphasia in a tactile personality type). L'Encephale, 1885, No. 5.

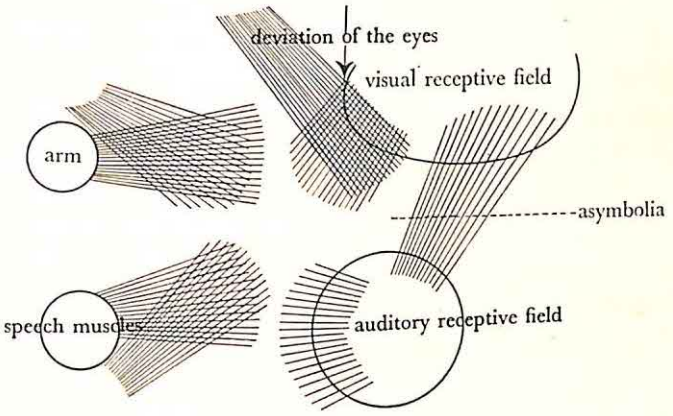
A second observation of this type caused C. S. Freund[1] to postulate the category of "optic aphasia". His patient showed difficulties in spontaneous speech and in naming objects very similar to those observed in sensory aphasia. The following is a sample of his reactions: he called a candlestick "spectacles", and on looking again he said, "It is for putting on, a top hat" and immediately afterwards: "It is a stearin light". If, however, he was allowed to take the object into his hands with his eyes closed he quickly found the correct name. His speech apparatus, therefore, was intact, but it failed when stimulated by way of visual object associations only, while it worked correctly when stimulated through tactile object associations. However, in Freund's case the effect of the disturbance of the object associations had a less severe effect than in Farges' case. Freund's patient deteriorated and later became completely word deaf. The post-mortem examination revealed lesions involving not only the visual but also the speech area.

The disabling effect which disturbances of the visual object associations may have on the speech function can be explained by the special importance that they assume in certain cases. In an individual whose thought processes depend largely on visual images, a peculiarity which according to Charcot is determined by individual predisposition, bilateral lesions in the visual cortex are bound to cause in addition disorders of the speech function which go far beyond what can be accounted for by the localization of the lesions. "*Aphasie chez une visuelle*" would have been a far more appropriate description for Farges' observation than "*aphasie chez une tactile*".

[1] C. S. Freund: *Ueber optische Aphasie und Seelenblindheit* (On optic aphasia and mind blindness). Arch. f. Psychiatrie, XX, 1889.

While agnostic aphasia was in these cases caused by a remote functional effect in the absence of an organic lesion of the speech apparatus itself, the verbal and asymbolic aphasias are manifestations of such a lesion. We shall endeavour as far as possible to differentiate the

FIG. 9



Anatomical schema of the area of the speech associations demonstrating how the appearance of speech centres is created. The auditory and visual receptive fields and the motor areas for the muscles serving articulation and writing are represented by circles. The association tracts connecting them with the interior of the speech area are represented by radiating fascicles. The area in which the latter are crossed by the corresponding fascicles from the other hemisphere becomes a centre for the respective associative element when the tracts are cut off from the fields represented by circles. The crossed connections of the auditory receptive field have been omitted from the schema to avoid confusion, and also because of the uncertainty of the connections between the auditory receptive field and the sensory speech centre. The separation of the connections with the visual receptive field into two fascicles is based on the consideration that the eye movements play an important part in the associations contributing to the act of reading.

functional from the topographical factors in the analysis of these speech disorders.

We have designed a schema (Fig. 9) which is meant to illustrate the relations between the various elements of speech associations without taking into account anatomical details. In this schema the circles do not represent the so-called speech centres but show the

receptive and motor cortical fields between which the speech associations take place. The parts of the speech area bordering on these cortical fields acquire the significance of centres by virtue of their crossed connections with the other hemisphere: those connected with the cortical fields for the hand, the speech muscles and the optic nerve can be seen in the schema. It follows that in the case of a verbal aphasia, three symptoms can be accounted for by the localization of the lesion: if the latter is situated within the "speech centres", the following functions will be impaired: (1) transmission of stimuli to the tracts serving the speech muscles and (2) to the tract serving the muscles employed in writing, and (3) recognition of letters. The resulting disorder is a typical motor aphasia with agraphia and with alexia for letters. The further the lesion moves towards the centre of the speech region the less likely it is to cut off any single element of the speech associations, and the more the features of the aphasia will depend on functional factors to which the speech apparatus is subject, independently of the site of the lesion. In verbal aphasia, therefore, only the loss of individual elements of the speech associations can be related to and explained by the localization of the lesion. It will help the diagnosis of the site of the lesion if the latter does not extend more centrally into the speech area, but rather into the adjoining receptive or motor cortical fields, i.e., if the motor aphasia is accompanied by a hemiplegia, or the alexia by a hemianopia.

The asymbolic aphasia may sometimes exist in a pure state resulting from a circumscribed lesion lying across the path of the association tracts. This had happened in Heubner's patient who presented an almost ideal example of the separation of the speech region from its associations by a vascular lesion encircling the auditory area which

is a nodal point of the speech region. Asymbolic speech disorder without complications, i.e., without disturbance of the word associations, may also result from a merely functional state of the speech apparatus as a whole; there are some indications that the link between word associations and object associations is the most easily exhaustible component of the speech function, its weakest point as it were. This was illustrated in an interesting paper by Pick[1] who had noted transient word deafness following epileptic fits. The patient observed by him showed an asymbolic speech disorder in the course of her recovery from the fit. Even before the understanding returned she was able to repeat words spoken to her.

The phenomenon of echolalia, i.e., the repetition of questions asked, appears to belong to the asymbolic disorders. In some of these cases, e.g., in those observed by Skwortzoff[2] (case X) and Fränkel[3] (quoted by Ballet), echolalia proved to be a means of overcoming the difficulties in relating the words perceived to the object associations by reinforcement of the word sounds. These patients failed to understand questions at first, but were able to understand and answer them after they had repeated them. This phenomenon also calls to mind Bastian's thesis that a speech centre, the function of which is impaired, loses first the ability to respond to "volitional" stimuli, while still able to react efficiently

[1] Pick: *Zur Localisation einseitiger Gehörshallucinationen, nebst Bemerkungen über transitorische Worttaubheit* (On the localization of unilateral auditory hallucinations with observations on transitory word deafness). Jahrb. f. Psychiatrie, VIII, 1889, and l. c. Arch. f. Psychiatrie, XXII, 1891.

[2] Skwortzoff: *De la cécité et de la surdité des mots dans l'aphasie* (On word blindness and word deafness in aphasia). Paris, 1881.

[3] Ballet: *Le langage interieur et les diverses formes de l'aphasie* (The inner language and the various forms of aphasia). Paris, 1886.

to sensory stimulation and in association with other speech centres. Every "volitional" excitation of the speech centres, however, involves the area of the auditory images and results in its stimulation by object associations. This goes to show that, while the so-called transcortical sensory aphasia can be caused by a lesion, there are functional factors which tend to produce a similar clinical picture.

The mixed asymbolic-verbal aphasia due to impairment of the auditory element of speech is more common than the pure asymbolia. As all other word associations are linked to the sound image, any substantial lesion of the speech area adjoining the auditory field is bound to result in disorder within the word associations themselves as well as in disturbances of their connections with the object associations. The resulting clinical picture is that of Wernicke's sensory aphasia which also comprises disturbances in the understanding of written language, in spontaneous speech and in repetition. The area in question is probably so large that smaller lesions may lead to clinical pictures in which either the verbal or the asymbolic disorder is more pronounced. Detailed anatomical knowledge of the points at which the various fibre tracts enter the auditory speech area would be necessary for more exact localization. Such knowledge is not available at present.

We may assume that the most important source for the association of symbols is the visual area of the cortex, because the visual images usually play the most important part among the object associations. If they are excluded, the speech region can still receive impulses from the remaining cortex, i.e., tactile, gustatory and other associations, and it can still be sufficiently stimulated for speech to be produced. This explains the marked poverty of words of special significance, especially of

nouns and adjectives, in patients with asymbolic-verbal aphasia, although spontaneous speech is not abolished even in the most pronounced cases. The words used are spoken mainly on visual stimulation. When stimulated by the other object associations entering the auditory area, the speech region produces a mutilated language, or it transmits all possible stimuli which do not require special object associations, such as particles or senseless syllables (jargon) to the motor pathway serving speech.

We remember that between the large area of the visual cortex and the cortical field of the acoustic nerve there pass not only the association tracts connecting word and object concepts, but also the tract which serves the understanding of visual letter images. A lesion of a certain localization can therefore, as the result of anatomical contiguity, cause alexia besides asymbolic aphasia. Clinical experience shows that such a combination of alexia with asymbolia of varying degree can actually be observed in cases of lesions of the angular and supramarginal gyrus. These symptoms need not, however, coexist, as has been pointed out before. As a rule, lesions of this region cause a purely verbal speech disorder which takes the form of alexia; for asymbolia to occur in addition, bilateral lesions of the visual cortex must be present. On the other hand, even a unilateral lesion suffices to cause asymbolia if situated closely to the auditory speech area, because of the connection of this "speech centre" with the visual association fibres from both hemispheres. The combination of asymbolia with word deafness, therefore, comes about more easily than that of asymbolia with alexia; the former requires only a unilateral lesion close to the auditory receptive cortical field, while for the latter to occur bilateral lesions are necessary which, however, need

not be situated close to the receptive cortical field.[1]

C. S. Freund, in designating the combined speech disorder under discussion as optic aphasia has, it seems to me, failed in separating the agnostic aphasic from the asymbolic component.[2]

This is as far as we can go in tracing the influence of the topographical factor of a lesion on the symptoms of speech disorder. Our main finding has been that this influence shows itself under the two following conditions: (1) when the lesion is situated in one of the speech centres in our sense, i.e., in the most peripheral areas of the speech region, and (2) when it puts this centre out of action completely. The lesion then results in a loss of one of the elements entering into the speech associations. In all other cases functional factors will play a part in addition to the topographical factor, and we have to decide which of the two conditions mentioned has been lacking. If, however, the lesion is situated in one of the

[1] It is probably not without significance that pure alexia, named "subcortical" by Wernicke, is so frequently found in cases with lesions of the angular and supramarginal gyrus. We remember that a lesion of the inferior part of the parietal lobe causes permanent deviation of both eyes, i.e., the kind of eye movement which in the act of reading becomes associated with the visual letter images.

[2] Siemerling, in his paper "*Ein Fall von sogenannter Seelenblindheit nebst anderweitigen cerebralen Symptomen*" (A case of so-called mind blindness, combined with other cerebral symptoms), Arch. f. Psychiatrie, XXI, 1890, thought that "it is possible to produce experimentally a condition resembling mind blindness merely by reducing visual acuity and by monochromasy". However, such an experimentally produced state is not entirely identical with the picture of visual agnosia. Besides, the patient tends to produce illusions because of his indistinct perceptions while the healthy subject simply feels undecided. Aphasic patients with alexia or word deafness also produce illusions. A patient described by Ross (l. c.) could read his newspaper for hours without understanding what he was reading; he was amazed at the nonsense put into the papers nowadays. Word deaf patients usually reply to a question because they believe they have understood it.

centres, without destroying it, this particular element of the speech associations will react as a whole with a change of its mode of function, and Bastian's modifications will come to the fore. If the lesion is situated in the interior of the speech area it will, however destructive, only cause such reductions of functions as I shall presently enumerate. They follow from the general organization of an apparatus of association. In this case the possible extent of the lesion is limited by the stipulation that it must not touch a centre anywhere.

In assessing the functions of the speech apparatus under pathological conditions we are adopting as a guiding principle Hughlings Jackson's doctrine that all these modes of reaction represent instances of functional retrogression (dis-involution) of a highly organized apparatus, and therefore correspond to earlier states of its functional development. This means that under all circumstances an arrangement of associations which, having been acquired later, belongs to a higher level of functioning, will be lost, while an earlier and simpler one will be preserved. From this point of view, a great number of aphasic phenomena can be explained.

(1) The loss, through damage to the speech apparatus, of new languages acquired as super-associations, while the mother tongue is preserved. Next, the nature of the speech remnants in motor aphasia which are so frequently only "yes" and "no" and other words in use since the beginning of speech development.

(2) Furthermore, it can be stated that the most frequently practised associations are most likely to resist destruction. This is why patients suffering from agraphia are still capable of writing their names, if anything at all, just as illiterates are capable of writing nothing else. (In motor aphasia, however, the patient's own name is not spared, nor is this to be expected as we only rarely

pronounce our names.) The above statement implies that the patient's occupation may conspicuously manifest itself in his symptoms; e.g., Hammond reported the case of a ship's captain with asymbolic aphasia who could name things only by nouns referring to naval objects. Also, whole functions of speech will, according to this principle, prove more or less resistant in the case of a lesion. I am inclined to agree with Marcé[1] who attributed the fact that in an aphasic barrister the ability to write to dictation was hardly impaired, to the patient's special skill in taking down information. It is to be expected that the manifestations of aphasia will be different in the highly educated from those in the illiterate. This has to be studied in each individual case.

(3) On the other hand, a rare product of speech may prove highly resistant if it had acquired great force by being associated with great intensity. I referred to such instances earlier when discussing speech remnants ("recurrent utterances") which, according to Hughlings Jackson, are last words.

(4) It is also noteworthy that series of words are better preserved than single ones, and that words remain the more easily available the more widespread their associations are. The former rule applies to series such as successive numbers, days of the week, months, etc. Grashey's patient was unable to state a certain number directly, but he got round this difficulty by counting from the beginning until he arrived at the requested number. Sometimes the whole series of associations can be recited, but not one particular part in isolation. Kussmaul a.o. have reported numerous instances of this type. It even happens that people who are incapable of uttering a single word spontaneously, are able to sing a song perfectly correctly.

[1] Quoted by Bastian. *On the various forms etc.*, 1869.

(5) In the speech disorders resulting from asymbolia, it is obvious that the words most likely to be lost are those with the most specific meaning, i.e., those which can be elicited by only few and definite associations. Names of persons are forgotten most easily, even in the physiological amnesia; in asymbolia nouns are affected first, adjectives and verbs later.[1]

(6) The effects of fatigue after prolonged associative activities, of the reduction in the duration of the sensory impressions, and of fluctuating and erratic attention are important for the individual features of a speech disorder, but too obvious to require special proof.

Most of the factors enumerated here are inherent in the general properties of an apparatus equipped for association; similar factors play a part in the functions of other regions of the brain under pathological conditions. Perhaps the most striking counterpart of speech regression is the total loss of all cortical associations acquired up to a certain early period of life, occasionally observed after head injury.

The three levels of reduced functional efficiency of the speech centres postulated by Bastian, have been mentioned several times already. We can accept them, although we reject the notion of physiological speech centres. We prefer the formulation that the visual, auditory and kinaesthetic parts of the speech apparatus are still able to function under certain specifiable conditions. We also have to realize that Bastian's modifications apply mainly to incompletely destructive lesions, especially of the "speech centres": if the lesion does not affect all elements of one particular speech function, such as is the case when the damage is situated in the nodal

[1] See Broadbent: *A case of peculiar affection of speech with commentary.* Brain, I, 1878-1879, p. 494.

points, the activities of the intact portion of the nervous tissue will compensate for that of the damaged part and cover up the defect. A statement such as this implies, of course, that no single nerve fibre and cell can serve one single function of speech only, but that the conditions are in fact more complicated than this.

Bastian's modifications represent, in a sense, also levels of disinvolution, i.e., of functional retrogression. I regard it, however, as profitable to discuss them for each element of the speech associations.

(1) The acoustic element is the only one which responds to three different types of stimulation. The one called "volitional" by Bastian, consists of stimulation from the object associations, or more exactly from the activities of the rest of the cortex. This type of association is liable to break down as the result of comparatively slight lesions in the auditory speech centre causing a partial asymbolic aphasia. The latter manifests itself by impairment of spontaneous speech and of the ability to name objects. In the mildest form there is only some difficulty in finding nouns of strictly limited significance and with a small range of associations.

The associative activity of the acoustic element is the central part of the whole speech function. Grashey's and Graves' cases illustrate a disorder of voluntary speech with intact ability of association with the visual element. I have been unable to find examples of failure of the acoustic element to associate spontaneously, while still functioning on direct stimulation. Such a state would probably imply a complete loss of function, as the activity of the acoustic centre consists in association and not in transmission to an efferent tract. On the other hand, it may happen that the acoustic element may still be able to produce verbal associations on peripheral stimulation, but no longer associations of symbols. Such a disorder

would again manifest itself as asymbolia, i.e., as Lichtheim's transcortical sensory aphasia. From this we are inclined to infer that the latter type of speech disorder can be caused by a lesion in the auditory centre as well as by one situated between the latter and the visual receptive cortical field. In the former case it would be caused through functional, in the latter through topographical factors.

It seems that loss of excitability of the acoustic element, which manifests itself as word deafness, is invariably to be interpreted as a local symptom. The only exception is the rather obscure group of cases mentioned by Arnaud,[1] which might suitably be called "partial word deafness". It is essential for their understanding that they invariably show a considerable degree of ordinary bilateral deafness. These patients speak quite correctly, but they can understand only with difficulty and have to be spoken to slowly and distinctly. Under this condition their comprehension is immediate and complete, which argues against the assumption of a lesion in the so-called auditory speech centre. The only difference between the behaviour of these patients and that of ordinary deaf people lies in the fact that the latter can understand, i.e., associate, while listening, whereas the former begin to understand only when the strength of the peripheral stimulus is above certain thresholds.

Understanding of spoken words is probably not to be regarded as simple transmission from the acoustic elements to the object association; it rather seems that in listening to speech with understanding, the function of verbal association is stimulated from the acoustic elements at the same time, so that we more or less repeat to

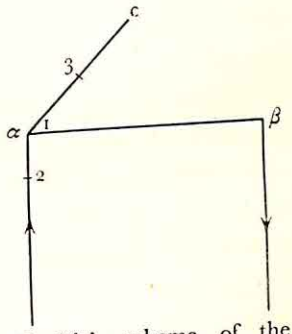
[1] Arnaud: *Contribution à l'étude de la surdité verbale* (A contribution to the study of the pure word deafness). Arch. de Neurol., Mars 1877.

ourselves the words heard, thus supporting our understanding with the help of kinaesthetic impressions. A higher measure of attention in listening will entail a higher degree of transmission of speech heard on to the tract serving the motor execution of language. One may suppose echolalia to occur if there is an obstacle in the connection with the object associations; under these circumstances the whole of the excitation may be discharged by way of an even stronger, i.e., an audible repetition of words heard.

(2) The visual element is not directly linked to the object associations, as our letters represent sounds and do not symbolize concepts like those of certain other peoples; we do not therefore need to consider spontaneous stimulation here. This element is activated mainly by peripheral stimulation and, in the case of spontaneous writing, by mere association with other elements of the word concept. Inability to recognize letters is the only manifestation of impairment of the visual element, as "reading" is a much more complicated function which can be impaired by a great variety of lesions. Here the exceptional case of an element no longer responding to peripheral but still to associative stimulation can apparently occur: there are patients who are unable to recognize letters but can write well. Wernicke calls this disability subcortical alexia and explains it with the localization of the lesion. He differentiates three disorders of reading whereby the word concept (C) is intact (Fig. 10): (1) Cortical alexia characterized by loss of reading and writing; (2) Subcortical alexia; loss of the ability to read; writing is unimpaired except for inability to copy; (3) Transcortical alexia; there is loss of reading and writing, but the ability to copy printed or written words mechanically is preserved.

There is a simple objection to this schema for the disorders of letter reading. If in subcortical alexia the lesion is situated in the peripheral fibre tract leading to α, no impression of the letters presented can reach the cortex; they are not seen and therefore cannot be copied, unless each letter is normally seen via two pathways, the one perceiving it as an ordinary visual object and the other as speech symbol. The same objection could not be raised for the so-called subcortical word deafness, because the word that is not heard is not repeated either. But the letter that is not recognized can be copied, and therefore the failure of recognition cannot be due to a lesion before α; we are not dealing with a disorder of perception but with one of association. Wernicke, it is true, distinguished between "copying" and "drawing from a model", thus trying to save his theory. However, I believe that interruption before α must impede both motor functions, unless we assume that every letter image reaches the brain by two different peripheral tracts, as an ordinary object and as an object for speech.[1] Copying differs from drawing from a model only by the degree of facilitation derived from the comprehension of the model; otherwise they are identical and use the

FIG. 10



Wernicke's schema of the disorders of reading. (Die neueren Arbeiten über die Aphasie (Recent studies of aphasia) Fortschritte der Medicin, 1886, p. 464.) *a* the visual image of letter, word or numerals; *β* the motor centre of the writing movements; $c=a+b$ the word concept.

[1] It could be argued that this does in fact happen, because this type of alexia is usually accompanied by right-sided hemianopia, the letter being perceived as object for speech by the left and as ordinary object by the right hemisphere. However, if this were so, every right-sided hemianopia would have to be accompanied by alexia, which is not the case.

same pathway. Everybody requires a high degree of attention for copying incomprehensible symbols; this is as a rule difficult to obtain in aphasics. There is, however, an alternative explanation: copying consists in a transposition of printed letters into letters of script. This transposition is possible because we learn to read, but not to write, normal print or italics, and it makes no difference whether or not the words read are understood. A child patient observed by Bernard (Obs. V) showed a remarkable facility in faultlessly carrying out this transposition in copying though he was quite unable to read what he had copied.

In my opinion the so-called subcortical alexia calls for a different explanation. In writing as well as in speaking we receive kinaesthetic impressions from the movements carried out by the muscles involved. However, the impressions coming from the hand are more distinct and intensive than those coming from the speech muscles, either because we are used to attributing great value to the perceptions of the hand also in relation to functions other than writing, or because they are associated with visual impressions: we can see ourselves writing but not speaking. Therefore we are able to write directly from the sound images with the aid of kinaesthetic impressions without depending on the visual element.

The lesion underlying subcortical alexia can be assumed to be situated on the periphery of the speech region as it is so frequently accompanied by hemianopia. In this type of aphasia, therefore, the whole motor apparatus may be intact and writing is possible through direct associations with the word sounds. In some cases of subcortical alexia reading is aided by writing as mentioned earlier; the letter images incapable of direct association with the acoustic element, are nevertheless

associated with it by means of the kinaesthetic impressions aroused in the process of "drawing from a model", and in this way they are recognized.

Almost all authors who reported instances of mixed aphasia with writing and reading disorders state that in these cases the degree of the writing disorder was proportionate to the motor impairment of speech rather than to the disorder of reading. This would be impossible if writing had not become independent of the letter images after some practice. Self observation, I believe, also shows that in writing spontaneously one does not rely on the visual element, except when writing foreign words, proper names and words which one has learned by way of reading only.[1]

Impairment in the recognition of letters naturally implies inability to read. However, it is possible for a reading disorder to be present without loss of the ability to recognize letters. This may result from a variety of lesions and conditions such as can be readily understood from earlier remarks about the intricate processes of associations which enter into the act of reading. A reading disability may be no more than the result of increased exhaustibility of the visual function, whereby motor aphasia or disturbance of auditory associations need not be present. This applied to a case of Bertholle mentioned by Bernard, and to the so-called dyslexia described by Berlin.[2] In such a case the failure to read

[1] I believe that some physiological and individual peculiarities of memory can be explained by the changing role of its individual elements. One may have a very good memory yet be unable to retain proper names and numbers. Individuals who excel in remembering names and numbers belong to the visual type, i.e., they have a predilection for recalling the visual images of objects even if they think in sound images.

[2] Berlin: *Eine besondere Art der Wortblindheit: Dyslexie* (A special type of word blindness: dyslexia). 1887.

will be preceded by an attempt at spelling which may at first be successful. This may mean that the impaired visual element be still capable of the simpler function of associating visual images with the acoustic and kinaesthetic elements while no longer able to cope with the numerous repetitions and the proper arrangement of this activity which, if performed with a certain speed, makes up the process of reading. This is an instance of the loss of a complicated function while the simpler one is preserved.

Alexia may also be the result of damage to the motor or auditory element of speech; this naturally deprives it of diagnostic significance. I think it can be maintained that generally motor aphasia abolishes the understanding of written language as well as the so-called mechanical reading because the understanding of read material is accomplished only after the transmission of the stimulus from the visual to the motor elements through the association of the latter with auditory impressions. In cases of auditory impairment, however, as well as of asymbolia, the purely mechanical type of reading may be preserved. The explanation of reading disorders, which I do not intend to discuss in detail here, presents certain difficulties which cannot be disposed of by reference to the localization of the lesion, nor by assuming the familiar functional changes. In complicated cases one or the other part of the reading function remains intact, depending on the specific elements of association having been spared to a greater or lesser degree in individual patients.

(3) The motor element, i.e., innervation impressions and kinaesthetic images, presents fewer difficulties. We assume that volitional and associative stimulation of this element usually coincide as spontaneous speech is activated via the sound images. The so-called stimula-

tion from the periphery is also an association, being activated either through the auditory element, as in repetition of spoken words, or through the visual element, as in reading aloud. It seems that occasionally the latter succeeds, but not the former, and vice versa. In the so-called transcortical motor aphasia we have an instance of the motor element still being capable of stimulation by peripheral association, but failing to respond to volitional association.

Nevertheless, the understanding of motor aphasia, which has been known better and for a longer time than any other type, offers more difficulties than one would expect. We have already referred to the uncertainty as to whether in motor aphasia the function of symbolic association, i.e., the volitional stimulation of sound images, is really intact. If the opposite could be established it would mean that the loss of the motor element impairs the function of the auditory element in the same way as we have long known it to happen in reverse. Furthermore, there are the inexplicable cases of motor aphasia with letter blindness which one can hardly attribute to a chance coincidence.[1] Finally, it has never been satisfactorily explained why cases of total loss of motor speech are so common, while reduction of speech to half or a third never occurs. Cases of the latter type invariably turn out to be sensory aphasias on closer analysis. It seems that a lesion capable of disturbing the motor function of speech destroys it completely in most cases, apart from the well-known scanty remnants.

There is, as it were, no paresis, but only paralysis. Also, the failure of most cases of motor aphasia to improve, deserves attention. This is in striking contrast to the rapid and complete return of speech in other cases. It is

[1] Bernard has reported a case of this type. l. c. p. 125.

hardly necessary to point out that speechlessness in the first days after the onset of an illness has no diagnostic significance. It may occur irrespective of the site of the lesion, and is obviously caused by the shock to the apparatus which had previously been working with all its resources.

(4) I do not intend to enter into a similar discussion of the cheiro-motor element. Some important considerations regarding it have been advanced earlier, when the function of the visual component of speech was discussed.

I now have to consider an interesting and significant idea introduced into the theory of aphasia by Charcot.[1] Its acceptance would impose considerable limitations on our hypotheses in this field. We have assumed that in spite of equal potentialities of the associations between the various elements of the speech function, nevertheless certain kinds of associations have preference over the rest; in the speech disorders, therefore, not all and sundry, but only a limited number of associations between the elements of speech need be taken into account. We have postulated that they are those which play a leading part in the learning of speech. In Charcot's view, no such general rule of preference of routes of associations exists; all links between the elements of speech appear at first to be endowed with equal functional rights, and it is left to individual practice or organization to make one or the other element of speech the central co-ordinating factor for the rest. According to this theory one individual speaks, writes and reads predominantly or exclusively with the help of kinaesthetic sensory impressions, while another may employ the visual element for the same purpose, etc. This would rule out an over-all dependence

[1] Charcot: *New Lectures*, 1886. See also the papers of his pupils Ballet, Bernard and Marie.

of the function of speech association on the contribution of the acoustic element.

It can easily be seen that identical lesions would result in different speech disorders, if such a relationship exists. A "motor" speaker could suffer a lesion of the acoustic or visual element with hardly any ill-effect, while damage to his motor element would deprive him of almost all speech functions in addition to the motor one. Damage to the visual element would render a "visual" speaker not only letter blind, but nearly or completely incapable of using his speech apparatus at all. The diagnosis of aphasia would be liable to the grossest errors if inferences concerning the site and extent of the lesions were drawn from the loss of functions sustained, before the individual's preference for a single associative element had first been ascertained; such knowledge could be obtained only in the most exceptional cases.

Nobody has so far wanted to reject Charcot's approach completely. However, its significance for the theory of aphasia is open to doubt. Extreme claims, such as those advanced by Stricker[1] for the paramount importance of the motor element of speech, have been refuted by Bastian with the remark that he was waiting until he was shown a case of a person who had been made word deaf through destruction of Broca's area. I believe that the study of the speech disorders has so far provided no reason for attributing any great importance to Charcot's speculations regarding the main aphasic symptoms. However, the possibility cannot be excluded that as long as the speech apparatus is in possession of all its resources, such a habitual preference for one or the other speech association may exist, but that in case of illness, i.e., of lowering of associative activity, the pre-eminence of the

[1] Stricker: *Studie über die Sprachvorstellungen* (Studies of the speech concepts) 1880.

first used lines of associations is re-established. It would certainly be wrong to dismiss Charcot's idea completely and to allow oneself to be misled into a schematic rigidity in the interpretation of the speech disorders. "Different amounts of nervous arrangements in different positions are destroyed with different rapidity in different persons", says Hughlings Jackson.

Summary and Results

We can now survey the route we have travelled in this treatise. Our starting point was Broca's discovery which for the first time related a certain form of speech disorder, i.e., the motor aphasia for which he proposed the term aphemia, to lesion of a certain area of the cerebral cortex. When Wernicke repeated this feat for another type of aphasia, the way was open for explaining differences of speech disorders by differences of localization. Wernicke distinguished sharply between speech centres and fibre tracts; he characterized centres as storing places of impressions and postulated a conduction aphasia (commissural aphasia) in addition to the two main forms of speech disorder mentioned above. Lichtheim, in considering the possible connections of the speech centres with the rest of the cortex, increased the number of conduction aphasias and attempted to interpret other varieties of speech disorders as subcortical and transcortical aphasias. Thus the contrast between central aphasias and conduction aphasias was assumed to be the key for the understanding of the speech disorders. On the other hand, Grashey, in his theory of the amnesias, completely discarded the basic explanation by localization and, in an ingenious analysis, attributed a certain type of disorder to alteration of a functional constant in the apparatus of speech. According to him, disturbances of speech fall into two groups: the one due to localized

lesions, and the amnesias due to functional change not localized anywhere in particular. We set out with the intention of examining whether the principle of localization could really offer as much for the explanation of the aphasias as has been claimed, and whether one is justified in differentiating between centre and pathways of speech and between the respective types of speech disorders. We first analysed Wernicke's conduction aphasia, and found that according to his schema it ought to have different features from those he had attributed to it, features such as nobody is ever likely to find in reality. We then turned to one of Lichtheim's conduction aphasias, the so-called transcortical motor aphasia, and we established with the help of several post-mortem findings that it was due to a lesion in the motor and sensory centres themselves and not in the fibre tracts, and that the pathway the lesion of which Lichtheim regarded as the cause of this aphasia, probably did not exist at all. In the course of our study we also discussed other subcortical and transcortical aphasias, and we found on every occasion that the lesions had been situated in the cortex itself. Only to the transcortical sensory aphasia, for which we proposed the name asymbolia, did we have to concede a specific localization. Our views were strongly supported by a case reported by Heubner. However, the fact that cortical lesions of the same localization could cause such different clinical pictures called for an explanation. We put forward the assumption that the so-called speech centres as wholes reacted to partial damage with a modification of function. Regarding the kind of modification, we followed Bastian who recognized three pathological conditions of a centre: (1) Absence of excitability to volitional stimulation, with preservation of excitability through association and to sensory stimuli. (2) Loss of excitability except

by sensory stimuli. (3) Complete loss of excitability.

While thus resorting to functional factors in the explanation of the so-called cortical aphasias, we found ourselves unable to accept as satisfactory Grashey's attempt to explain a case of amnesia by functional changes only. We proved that the topographical factor, too, had been of importance in this case and we explained its clinical features by referring, in addition, to one of Bastian's modifications.

Having rejected the differentiation between centre and conduction aphasia and between aphasias and amnesias, it was incumbent on us to evolve another conception of the organization of the speech apparatus, and to state how in our view topographical and functional factors manifested themselves in the disorders of this apparatus. After a critical digression to Meynert's doctrine of the organization of the brain and of the localization of concepts in the cortex, we successively rejected the following assumptions: that the impressions with which the speech function works could be localized separately from the process by which they were being associated; that association was carried out by way of subcortical fibre tracts; and that between well-defined centres there extended a functionless region waiting to be occupied by new acquisitions. Our concept of the structure of the speech apparatus was based on the observation that the so-called speech centres border externally (peripherally) on parts of the cortex which are important for the speech function, while interiorly (centrally) they enclose a region not covered by localization which probably also belongs to the speech area. The apparatus of speech therefore presented itself to us as a continuous cortical area in the left hemisphere extending between the terminations of the acoustic and optic nerves and the origins of the motor tracts for the

muscles serving articulation and arm movements. The necessarily ill-defined parts of the speech region which border on these receptive and motor cortical fields, have acquired the significance of speech centres from the point of view of morbid anatomy but not in respect of normal function; their lesions cut off one of the elements of speech association from its connections with the others. A lesion situated centrally in the speech region can no longer have this effect. We have made the additional assumption that this speech region is connected with the cortical fields of the right hemisphere via the corpus callosum, and that the connecting crossed fibre tracts also enter into the most peripheral parts of the speech region, i.e., the speech centres. Within the speech region we recognized conduction aphasias only, i.e., aphasias due to interruption of associations, and we denied any subcortical lesion the ability to produce aphasia, because there is only one pathway to the periphery, i.e., the tract which runs through the knee of the internal capsule, and its lesion causes anarthria.

In considering the effects of lesions on this apparatus we found that they could result in three types of aphasia: (1) purely verbal, (2) asymbolic and (3) agnostic aphasia. The discovery of the last named was a necessary corollary of our theory according to which destruction of both cortical areas for one of the elements involved must have the same effect as unilateral destruction of the nodal point for this element.

From the psychological point of view we recognized the word as a complex of concepts (impressions, images) which through its sensory part (its auditory component) is connected with the complex of object associations. We defined verbal aphasia as a disturbance within the word complex, asymbolic aphasia as a separation of the latter from the object associations, and agnostic aphasia

as a purely functional disorder of the speech apparatus.

Finally, the following factors have proved to be decisive for the effect of lesions on the speech apparatus so organized: the degree of destructiveness of the lesion, and its situation relative to the interior and the periphery of the speech region. If situated on its periphery, i.e., in one of the so-called speech centres, its symptoms are related to its localization; depending on whether it causes complete or incomplete destruction it either results in a loss of only one of the elements of speech associations, or it alters the functional state of this element in a way described as Bastian's modifications. If the lesion is situated centrally in the speech region the whole apparatus of speech suffers functional disturbances such as arise from its character as an instrument of association, and which we have attempted to enumerate.

Conclusions

I am well aware that the considerations set out in this book must leave a feeling of dissatisfaction in the reader's mind. I have endeavoured to demolish a convenient and attractive theory of the aphasias, and having succeeded in this, I have been able to put into its place something less obvious and less complete. I only hope that the theory I have proposed will do more justice to the facts and will expose the real difficulties better than the one I have rejected. It is with a clear exposition of the problems that the elucidation of a scientific subject begins. I should like to formulate the essence of my views briefly in a few sentences. Previous writers on aphasia who knew only of one cortical area with a special relation to speech disorder, found themselves compelled by the incompleteness of their knowledge to seek for an explanation of the variety of speech disorders in functional peculiarities of the apparatus of speech. After

Wernicke had discovered the relationship of the area called after him to sensory aphasia, the hope was bound to arise that this variety could be fully understood from the circumstances of localization. It appears to us, however, that the significance of the factor of localization for aphasia has been overrated, and that we should be well advised once again to concern ourselves with the functional states of the apparatus of speech.

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